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EDITORIAL

WE greet 1937 with a sense of security and satisfaction, based in no small degree on what has transpired during the year just ended. From all indications economic conditions are better and good times truly are at hand.

We have on hand many excellent papers covering a variety of subjects. Our policy of issuing special numbers has met with wide approval and will be continued.

The special February number is devoted to Obstetrics and is edited by Dr. H. J. Stander, Professor of Obstetrics and Gynecology at the Cornell University Medical College. This issue will prove to be an outstanding achievement. The articles are practical and decidedly worth while, and anyone doing midwifery will find them invaluable. The April number, which will be devoted to Office and Minor Surgery, will contain about sixty articles, each one to the point and aimed to help the physician in his every day work.

With this January number we include as an added feature, Dr. Henry C. Falk's monograph, "Practical Clinical Gynecology," which will appear in several issues. From time to time, other monographs on modern, practical topics will be published.

As heretofore, all advertising matter will be censored and only those decidedly in good taste and backed by reputable concerns accepted.

Many members of our editorial board and other physicians have given unstintingly of their time in reading and judging manuscripts, and from these contacts we have gained much in constructive criticism and knowledge.

To the members of our editorial board and to those friends who have been helping us quietly, and to the many subscribers who have written letters offering criticisms and suggestions, we wish to express our sincere appreciation.

We begin the tenth year of our editorship with a feeling that the dark, uncertain days have vanished in yesterday's mists, and that 1937 portends finer and better things for all of us.

T. S. W.

INTRACRANIAL ANEURYSMS

REPORT OF 13 CASES*

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THE medical literature concerning aneurysms of the cerebral vessels is very abundant, and no attempt will be made in this paper to review the previous work. Among the larger series of reported cases, especial reference may be made to those of Fearnsides and Beadles.

This paper attempts to review a series of cases with particular reference to the medicolegal aspect of intracranial aneurysms, a phase of the subject which has been given little attention in earlier studies. Although the cases in this communication are grouped according to etiology, certain of their aspects will be discussed briefly in the light of medicolegal interest. Examples of intracranial aneurysms occur not infrequently in which the etiology is obscure or inconclusive, even after careful postmortem study. The rupture of such aneurysms may be of especial importance from a medicolegal aspect.

The 13 cases of this series are readily classifiable into five categories from an etiologic viewpoint. Seven of them are intimately associated with arteriosclerosis of the cerebral vessels. Hypertension is usually an added factor either in the production of the aneurysm or in its rupture. For purposes of brevity, this group is listed as the arteriosclerotic group.

The second category comprises 2 cases with a well established history of marked hypertension and only slight evidence of atheromatous change in the cerebral vessels. In both instances the aneurysm occurred at the bifurcation of vessels forming the circle of Willis, a point which represents a natural weakness in the anatomic structure of vessels. Of the three factors concerned in the etiology of the aneurysms in these 2 cases, namely, hypertension, atherosclerosis and a *locus minoris resistentiae*, the hypertension seems to be the most important. This group is referred to as the hypertensive group.

Two cases fall naturally into the group of syphilitic aneurysms. This etiology of the aneurysms is well substantiated, both clinically and anatomically.

Case XII in the series presents some difficulty in classification on a single etiologic basis. A history of trauma prior to the rupture of the aneurysm strongly suggests this as the important etiologic factor. The age of the patient, two years, suggests a congenital basis for the lesion. Either or both of these factors may be the basis for the aneurysm.

Case XIII of the series defies classification. As will be commented later, the absence of any clinical history as well as the absence of any vascular disease makes a definite categorization impossible.

Various writers have affirmed that a certain diagnosis of intracranial aneurysm cannot be made except from necropsy findings. Others feel that the clinical signs may lead one to suspect the presence of an aneurysm of the cerebral vessels. Still others feel that in many cases the signs and symptoms are so definite that the diagnosis before death is easy and quite certain. It is obvious, however, that many intracranial aneurysms give rise to so little clinical evidence of their presence that a correct diagnosis cannot be made until the necropsy is performed.

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The series of intracranial aneurysms reported here consists of 13 cases which have been observed at the New Haven Hospital in 3600 consecutive autopsies, in about 80 per cent of which (2880 cases) the brain was examined. This incidence is somewhat lower than that reported by many writers, yet it may be seen that aneurysms of cerebral vessels are not extremely rare. It is likely, also, that more cases would have been found if the brain had been examined in every case, since it is well known that cerebral aneurysms before their rupture often produce no symptoms whatever.

REPORT OF CASES

ARTERIOSCLEROTIC GROUP

Case 1. A white female, aged sixty-two years, had a long history of paroxysmal headaches and hypertensive heart disease with symptoms of sudden severe pain in the head, loss of speech, confusion and retinal hemorrhages. Death occurred three days later. Autopsy revealed a rupture of an aneurysm of the right anterior communicating artery.

Clinical History. A. E. R., a white female, sixty-two years of age, was seen February 23, 1918, because of a sudden severe pain in the head. The patient had always been subject to paroxysmal headaches, often accompanied by visual disturbances and difficulty with speech. There had also been frequent attacks of numbness in the left arm and leg.

Physical examination on November 8, 1910, showed an obese woman of middle age with a moderately enlarged thyroid but with no signs of hyperthyroidism. The heart was considerably enlarged and there was a soft systolic murmur at the apex. The systolic blood pressure was 170 mm. of mercury. Under dietary treatment the patient lost weight and gained relief from dyspnea and palpitation.

Two years later there was noticed a systolic murmur in the aortic area, and the systolic blood pressure measured 205 mm. For the last three years of her life the patient complained very frequently of dyspnea and of pain which radiated down the left arm. She had numerous attacks resembling migraine. Repeated examination revealed the blood pressure to range between 180 systolic over 100 diastolic and 220 systolic over 115 diastolic.

The final illness began on February 23, 1918,

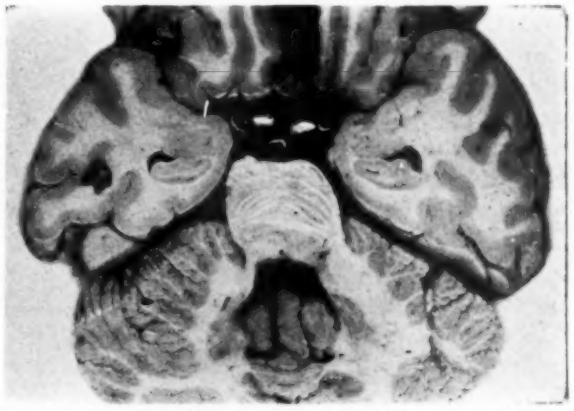

Fig. 1. Case 1. Photograph of horizontal section of brain through the aneurysm of the right anterior communicating artery.

three days before death, with sudden severe pain in the head, loss of speech and mental confusion. Consciousness was not lost. There was no paralysis or alteration in the reflexes. Vomiting occurred several times and the temperature rose to 101°F. in the evening. The next day the patient was still conscious but confused. Ophthalmoscopic examination of the left eye showed several large fresh retinal hemorrhages. The right fundus could not be seen clearly. On February 26, the patient seemed better in the morning but suddenly became worse and died in the afternoon.

Necropsy. The examination was performed by Dr. C. A. McKinlay, an abstract of whose findings follows. The heart weighed 335 gm. All the valves appeared essentially normal except the aortic, whose posterior and right lateral cusps were fused for a short distance from their insertions in the aorta. Numerous atheromatous plaques marred the intima of the aorta and formed a calcified ring at the insertion of the cusps. The renal vessels showed atheromatous changes and the parenchyma was extensively scarred. Many of the glomeruli were completely hyalinized.

Brain. A large, fresh hemorrhage filled the cisterns at the base of the brain and extended upward over the convex surfaces. A small amount of blood was present in the subdural space over each cerebral hemisphere. The hemorrhage seemed to emanate from an aneurysmal sac in the right anterior communicating artery (Fig. 1) and extended

through the cerebral parenchyma in the region of the third ventricle into the ventricular system which it completely filled.

On microscopic examination the wall of the aneurysm showed extensive atheromatous changes. The intima was elevated by plaques of cholesterin crystals and large, mononuclear, fat laden phagocytes. The elastic lamella of the media were completely absent in some parts and were fragmented in others. The smooth muscle was likewise fragmentary. Calcium salt deposits were scattered in the media. In the adventitia and periadventitial connective tissue large numbers of red blood cells were present as well as numerous phagocytes filled with blood pigment. The sac was roughly quadrilateral in shape and took origin from an artery which likewise showed extensive atheroma. The lumen of the aneurysm was partially filled with a recent thrombus.

COMMENT. The microscopic appearance of the aneurysm obviously points to arteriosclerosis as the etiologic factor. The age of the patient and the blood pressure substantiate this view. The only indication of a different etiology is speculative, and is based on the lesion of the aortic valve. An old endocarditis might conceivably have released an embolus and produced an embolic cerebral aneurysm, with ultimate healing. However, the fact that aortic valve lesions of this type are seen not uncommonly in arteriosclerosis, and the absence of any evidence of involvement of other valves by an inflammatory process, makes such a speculation extremely unlikely.

CASE II. A white female of fifty-four years presented a long history of attacks of headache and diplopia associated with vomiting. Her presented illness began with a sudden onset of severe headache and stupor. On admission there were stiff neck, left oculomotor paralysis and xanthochromic spinal fluid. The second attack was characterized by generalized convulsion and loss of consciousness. Death occurred twenty-nine days after first and ten days after second attack. Autopsy showed a rupture of an aneurysm of the left internal carotid artery with compression of the left oculomotor nerve.

Clinical History. M. M., a fifty-four year old white woman, was admitted to the New Haven Hospital, November 9, 1923, complaining of severe headache and pains over the eyes.

For about six months the patient had had frequent attacks of diplopia and headache with occasional vomiting. Twenty-nine days before death, while in bed, she was seized with a sharp pain in the epigastrium. On arising, she complained of severe pain in the back of the head, became faint and vomited. The following day, on admission to the hospital, she was stuporous and had stiffness of the neck and pain in the left eye. There was ptosis of the left eyelid and complete oculomotor paralysis on the left. The left pupil was larger than the right and did not react to light or accommodation. Both optic discs showed choking. All reflexes were equal and active, and there was no paralysis of the extremities. The blood pressure was 160 mm. systolic and 100 mm. diastolic.

The spinal fluid was blood tinged and xanthochromic and contained 350 cells per cu. mm., predominantly lymphocytes. The Wassermann reaction of the blood and spinal fluid was negative. The urine contained no albumin or sugar.

Ten days before death, she had a generalized convulsion, regaining consciousness in a short while, but she remained stuporous. Another convulsion occurred a few hours before death, following which she did not regain consciousness. Death occurred on November 22, 1923, nine days after admission.

Necropsy. The postmortem examination was made by Dr. G. B. Maurer. The body weighed 150 lb. (68.2 kg.) and measured 63 inches (158 cm.) in length. The heart weighed 310 gm.; one cusp of the aortic valve was adherent to the aorta by a small fan shaped adhesion. The other valve leaflets were normal. The coronary vessels showed considerable atheromatous change.

Brain. A small aneurysm 3 mm. in diameter, arose from the left internal carotid artery near the junction of the posterior communicating artery. To a perforation in the inferior surface of this aneurysm was attached a gray-red thrombus which was closely adherent to the left oculomotor nerve producing compression of it. The other arteries at the base of the brain were tortuous and showed marked atherosclerosis.

Microscopic preparations of the aneurysm prepared in serial showed that it opened into the internal carotid artery through a rather small orifice. The wall of the aneurysm was thin and irregular, while the wall of the internal carotid artery was thickened by atheromatous change and hyalinized connective tissue. The elastic tissue fibers of the artery were somewhat fragmented while none was present in the wall of the aneurysm. Fragments of a recent thrombus were adherent to the inner surface of the aneurysm.

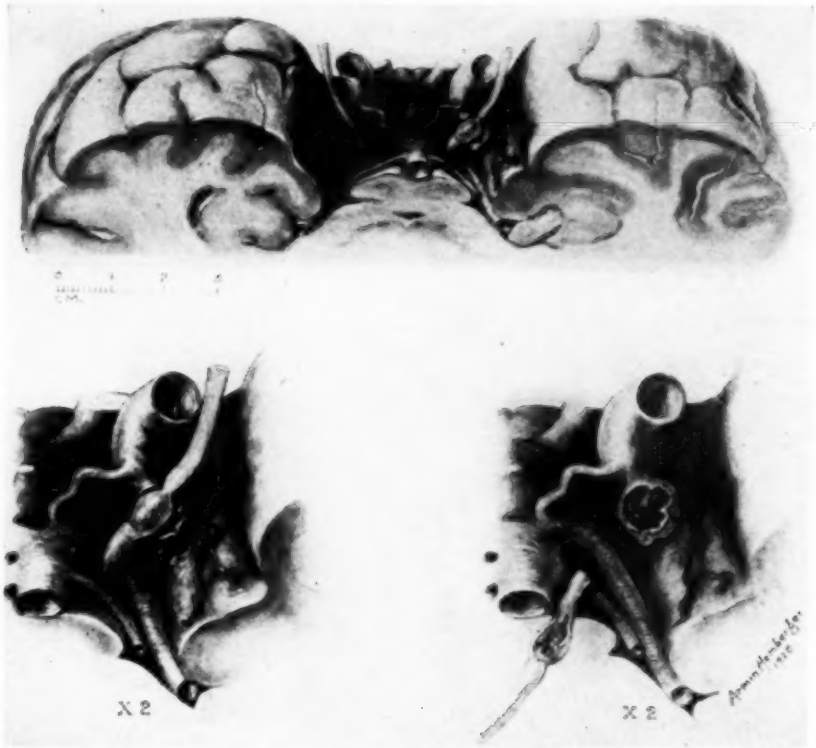

Fig. 2. Case II. Drawings to show ruptured aneurysm of left internal carotid artery and flattening of the left oculomotor nerve by the aneurysm.

COMMENT. Hypertension and rather marked atherosclerosis of the cerebral vessels are prominent factors in the formation of the aneurysm of the left internal carotid artery.

Case III. A white male of seventy years experienced sudden, severe stabbing pain below the left eye radiating into left side of head. He presented headache, diplopia, left oculomotor paralysis, loss of vision in left eye and hypertension. A second attack occurred eleven days later with severe back pain and death on the following day. Autopsy showed a rupture of an aneurysm of the left internal carotid artery with compression and degeneration of the left oculomotor nerve.

Clinical History. G. T., a white man, seventy years of age, was admitted to the New Haven Hospital, March 16, 1928, with complaints of weakness, headache and loss of vision in the left eye. The patient had always been in good health. On March 13, 1928, twelve days before death, while walking downstairs, he suddenly experienced lightning-like stabbing pain which passed from below his left eye into the left side of his head, and this was followed by a severe persistent headache. He did not lose consciousness; no paralysis was noted and no convulsion occurred. Within twenty-four hours he noted impairment of the vision of the left eye and a beginning ptosis of the left eyelid, which progressed to total blindness and paralysis in that eye during the next three days.

On admission to the hospital, the blood pressure was 190 mm. of mercury systolic and 112 mm. diastolic. The retinal vessels were sclerotic. The left disc showed some blurring of the margins but no swelling or hemorrhage. The third and fourth cranial nerves on the left side were paralyzed. The left pupil was widely dilated and did not react to light. The reaction

of the right eye was normal. There was a suggestion of a right facial weakness involving the lower musculature. The spinal fluid was bloody. The Wassermann reaction of the blood and spinal fluid was negative.

The first seven days of the patient's course in the hospital were uneventful. On March 23, he suddenly complained of feeling cold and of an intense pain in the back. He became unconscious and the respirations changed to the Cheyne-Stokes type. The right pupil was contracted and the left dilated; neither reacted to light. The neck was stiff. The abdominal, plantar and Achilles reflexes were absent. The temperature rose to 102°F., and he died the following day, twelve days after the initial cerebral accident.

Necropsy. The postmortem examination was made by Dr. H. M. Zimmerman. The lungs contained small zones of early focal pneumonia. The heart weighed 325 gms. It showed no dilatation, valvular lesions or endocardial changes. The aorta contained many atheromatous plaques, some of which were ulcerated. The peripheral arteries were all markedly thickened. The lumina of some of the branches of the renal artery and of the splenic artery were filled with thrombi and showed extravasation of blood in their walls, but the tissue supplied by these arteries did not appear infarcted.

Brain. An extensive fresh hemorrhage filled the subarachnoid space over the cerebrum, cerebellum and base of the brain, and also filled the ventricular system. There was a yellow atheromatous plaque in the left internal carotid artery 0.5 cm. proximal to its junction with the posterior communicating artery; just beyond this plaque there was a saccular aneurysm of the internal carotid artery, 4 to 5 mm. in diameter. The aneurysm had produced an indentation of the adjacent hippocampal gyrus and a flattening and thinning of the left oculomotor nerve (Fig. 2). A hemorrhage, localized to the vicinity of the left oculomotor nerve appeared older than the extensive subarachnoid and intraventricular hemorrhage. This older hemorrhage arose from a minute opening in the aneurysm which was filled with a friable gray-brown thrombus.

Microscopic preparations of the aneurysm prepared in serial showed the wall to be thin, fragmented, hyalinized and necrotic in various portions. Near an interruption in the continuity of the wall of the aneurysm there was a fresh hemorrhage which lay free in the subarachnoid space. Sections of the left oculomotor nerve stained with Heidenhain's hematoxylin showed extensive degeneration of the myelin sheaths.

COMMENT. Marked hypertension and extensive evidence of cerebral and peripheral arteriosclerosis played the important roles in the formation of the aneurysm in this instance.

CASE IV. A white female of fifty-eight years had sudden loss of consciousness and death occurred in four to six hours. At autopsy a ruptured aneurysm of the right anterior cerebral artery was found with massive intraventricular hemorrhage.

Clinical History. M. A. P., a white female of fifty-eight years, was admitted to the New Haven Hospital on May 24, 1932, in a comatose condition.

She had been observed in this hospital in 1921, at which time she had a large mass in the left upper quadrant of the abdomen. A definite diagnosis was not made, but Gaucher's splenomegaly was suspected. On the morning of admission the husband of the patient thought she did not look as well as usual, but she refused to have a physician called. When the patient's husband returned from work, a few hours later, the patient was lying on the floor in a semiconscious condition. She was admitted to the hospital about three hours later. The meager history which was obtained stated only that the patient had been "in poor health" for some time, with periods of "sinking spells" and severe headaches.

Physical examination showed a comatose, cyanotic woman having Cheyne-Stokes respirations. The pupils were widely dilated; the corneal reflexes were absent. The fundi showed numerous fresh hemorrhages and dilated vessels. The pulse rate was 120 and the heart sounds were obscured by many coarse rales and tracheal gurgles.

The periods of apnea became longer and the patient died thirty minutes after admission, about six hours after onset of the terminal episode.

Necropsy. The postmortem examination was made by Dr. J. C. Mendillo. The heart weighed 325 gm. and was not unusual. The abdominal mass noted eleven years previously

proved to be a Wilm's tumor of the left kidney. The right kidney was atrophied and its ureter was completely obstructed; the right ureteral orifice could not be probed. There was a leiomyoma of the stomach and multiple leiomyomata of the uterus.

Fig. 3. Case IV. Drawing of medial aspect of left hemisphere. The aneurysm of the right anterior cerebral artery is embedded in the left frontal lobe. The ventricular system is filled with clotted blood. Inset shows the laminated thrombus filling the aneurysm.

Brain. A subarachnoid hemorrhage was present in the Sylvian fissures, and over the base of the brain; freshly clotted blood filled the entire ventricular system. A large, firm mass which measured 3 cm. in greatest diameter, was attached to, and formed a part of, the right anterior cerebral artery (Fig. 3). This mass was embedded in the left cingulate gyrus and the corpus callosum. A large fresh blood clot attached to the posterior surface of the mass extended into the left lateral ventricle. On section the mass was identified as an aneurysm which was largely filled with a gray laminated thrombus. The wall of the aneurysm as well as the wall of the anterior cerebral artery from which it arose contained numerous atheromatous plaques. The posterior wall of the aneurysm was quite thin and for a distance of 4 to 5 mm. appeared to be entirely lacking.

Microscopically, the aneurysm showed extensive atheromatous change, with calcification in the media, and marked thickening and hyalinization of the intima. Considerable old blood pigment was present in the adventitia of the aneurysm. On either side of the point of rupture the aneurysmal wall was thin and necrotic.

An incidental finding in the brain was a meningeoma of the falx cerebri, which compressed the precentral gyri of either hemisphere.

COMMENT. Atherosclerosis involves the right anterior cerebral artery from which the aneurysm arose. Undoubtedly this degenerative lesion forms the basis for the formation of the aneurysm. Unfortunately, there is no clinical record of the blood pressure, but it seems fair to assume that this was elevated inasmuch as the right kidney was atrophied and its ureter obstructed at the same time that the left kidney was the seat of a Wilm's tumor. Even if an elevation of blood pressure did not contribute to the formation of the aneurysm, it may have played an important part in its rupture.

Case v. A white female of eighty-four years collapsed on arising from bed. Semiconsciousness, inability to speak, right hemiplegia and hypertension were present. Death six days later occurred. A thrombosed aneurysm of the left internal carotid artery and anomaly of the circle of Willis were found at autopsy.

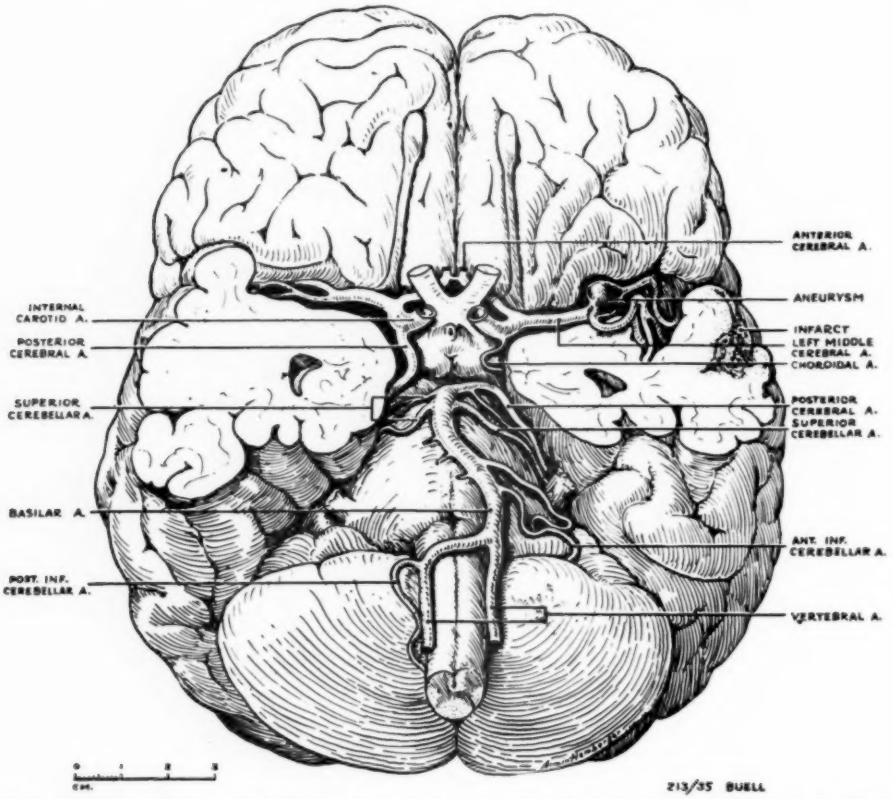

Fig. 4. Case v. Pen and ink drawing to show anomalous arrangement of the vessels composing the circle of Willis and the aneurysm of the left middle cerebral artery.

Clinical History. R. B., a white female, eighty-four years of age, was admitted to the New Haven Hospital on November 10, 1935, because of paralysis of the right side and stupor.

The patient had been well until a "shock" in 1933 which was accompanied by convulsions and followed by a period of coma. For three or four months she had some difficulty in speaking and since that time had had a poor memory. During the six months before her admission, she had dyspnea on exertion, attacks of cyanosis, and, more recently, edema of the ankles. On the morning of November 10, 1935, she fell when she attempted to arise from bed. She was not unconscious, but was unable to speak and apparently unable to move the right arm. She "slept deeply" during the morning, and was brought to the hospital about noon.

Physical examination showed a semiconscious condition and a right hemiplegia. The blood pressure was 180 mm. of mercury systolic and 120 mm. diastolic. The heart was enlarged and there was generalized arteriosclerosis. Many rales were heard in the lower lobes of both lungs.

The semiconscious condition deepened to complete coma. Her condition remained essentially the same until her death on November 15, 1935, six days after the onset of her symptoms.

Necropsy. The postmortem examination was made by Dr. H. Seely. There was marked generalized arteriosclerosis which involved especially the coronary arteries. The heart weighed 535 gm. The lumina of the coronary arteries were almost occluded by sclerotic plaques. The anterior descending branch of the left coronary was occluded by a thrombus and a rather fresh infarct of the myocardium of the left ventricle was present. The aorta contained numerous atheromatous and calcified plaques, some of which were ulcerated.

Brain. The vessels at the base of the brain were markedly thickened and sclerotic. At the

bifurcation of the left internal carotid artery into its three main divisions there was an aneurysm which measured 1 cm. in its greatest diameter (Fig. 4). The three branches arose directly from the aneurysmal sac which was filled with a thrombus. The portions of the brain supplied by these branches was softened and infarcted.

There was also present an anomaly of the circle of Willis. On the right side, the posterior communicating artery failed to join the basilar artery and continued directly as the right posterior cerebral artery. The left posterior cerebral artery arose directly from the basilar artery and no posterior communicating artery was present.

A large area of old softening was present in the posterior extremity of the left caudate nucleus. Large areas of the parietal lobe were infarcted as a result of the occlusion in the left middle cerebral artery.

Microscopically, the left middle cerebral artery showed extensive atheromatous changes and hyalinization of its walls. Near the aneurysm it was occluded by an organizing and canalized thrombus. The wall of the aneurysm showed similar atheromatous and hyaline changes. The sac contained clotted blood but this was evidently of postmortem origin.

COMMENT. The macroscopic and histologic appearance of the circle of Willis and particularly of the aneurysm of the left internal carotid artery conclusively show atherosclerosis to be the etiologic factor in the formation of the aneurysm. The age of the patient and her marked hypertension support this view. It is well to note the association of the aneurysm in this instance with the extensive anomaly in the circle of Willis. It is also of interest to note that this aneurysm failed to rupture, but rather produced cerebral infarction as the result of a thrombus which formed in its lumen.

CASE VI. A white male of thirty-seven years had frequent mild generalized headaches for several years, and slightly elevated blood pressure during preceding months, developed a sudden severe headache followed by stupor and coma. The spinal fluid was bloody. A second attack occurred thirteen days later with right hemiplegia and a third attack 14 days later was followed by death after a few hours. Rupture of an aneurysm of the left anterior cerebral artery was seen on autopsy.

Clinical History. W. McI., a thirty-seven year old white male, was admitted to the New Haven Hospital on November 18, 1935, complaining of a sudden severe headache.

For several years he had had mild generalized headaches several times a month. About twice monthly for fifteen years he had had attacks of precordial pain without other cardiac or gastric symptoms, which was diagnosed as "neurasthenia" at a distant hospital. Records from that institution showed that the blood pressure in 1929 was 120 mm. of mercury systolic and 80 mm. diastolic. In March, 1935, the pressure was 160 mm. systolic and 100 mm. diastolic. On the evening of November 17, 1935, he had a headache of the type which frequently occurred. At about 3:30 A.M. the following morning, immediately after intercourse, he "felt something snap" in his head. This was immediately followed by such a severe headache that he at once came to the hospital.

On admission the patient was rational but within a few hours became irrational and then semicomatose. The pupils were pinpoint and did not react to light. Kernig's sign was positive; the deep reflexes were hyperactive bilaterally; there was a positive Babinski bilaterally; the superficial reflexes were absent. The blood pressure was 160 mm. systolic and 90 mm. diastolic. The spinal fluid was grossly bloody. The Kahn reaction of the blood and the Wassermann reaction of the spinal fluid were negative.

Two days later he was again rational but lethargic. At that time the right pupil was larger than the left and both reacted to light; there was no strabismus; the neck was stiff; the patient was disoriented as to time; the headache continued. He improved during the next two weeks and again lapsed into coma. The neck was extended, the right side of the body flaccid, and the Babinski sign was positive on the right. Within a few days he again improved but neurological signs persisted. On December 14 the blood pressure rose to 180 mm. systolic and 100 mm. diastolic; the respirations changed to the Cheyne-Stokes type and the patient became comatose. Lumbar puncture showed fresh hemorrhage and greatly increased pressure. The patient died a few hours after the final attack, and twenty-seven days after the initial attack.

Necropsy. The postmortem examination was made by Dr. H. A. Weiner. The heart weighed 405 gm. and was not unusual. There

was very slight atheromatous change in the aorta. The lungs contained numerous small foci of aspiration pneumonia. The kidneys showed

In the region of the tuberal nuclei of the hypothalamus and adjacent periventricular gray matter there were numerous small recent

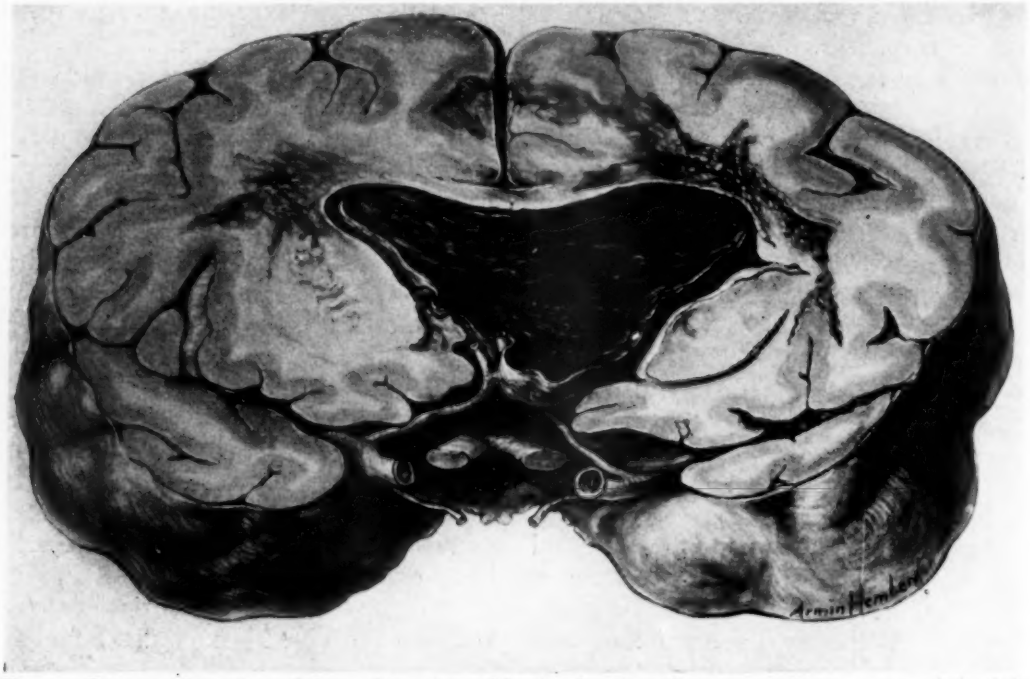

FIG. 5. Case VI. Drawing of frontal section of brain showing the ruptured aneurysm of the left anterior cerebral artery and the massive blood clot filling the ventricles.

some cloudy swelling.

Just proximal to the cardiac end of the stomach the esophagus was eroded and had ruptured, allowing gastric contents to flow into both pleural cavities. The pleura was also eroded but no evidence of an inflammatory reaction was present.

Brain. After fixation in formalin the brain weighed 1790 gm. The leptomeninges, particularly at the base of the brain, were filled with fresh clotted blood, which also filled and distended the entire ventricular system. The hemorrhage arose from a large aneurysmal sac attached to the right anterior cerebral artery (Fig. 5). This mass was about 1 cm. in its greatest diameter. The sac was filled with a laminated blood clot and the point of rupture of the sac was easily located.

Microscopically, both the aneurysm and the vessel from which it arose were the seat of quite extensive atheromatous change and hyalinization. The wall of the aneurysm was composed chiefly of hyalinized connective tissue. Both the aneurysmal wall and the wall of the right anterior cerebral artery contained old blood pigment.

hemorrhages which had destroyed many of the nerve cells.

COMMENT. Atherosclerotic changes associated with hypertension again point to the pathogenesis of the aneurysm and its final rupture in this instance. To be sure, the age of the patient, thirty-seven years, is somewhat disturbing in this connection. It should also be noted that there was a history of headaches in this patient long before any elevation of blood pressure was noted. Were it possible to link this history with the presence of an aneurysm already at that time, it would be necessary to seek elsewhere for the etiology of the hypertension.

CASE VII. A colored female of seventy-two years with a long history of hypertension, experienced sudden severe pain above the right eye followed by stupor, paralysis of the right oculomotor nerve and left hemiplegia. Death occurred five days after onset. A similar episode four years previously was followed by semi-

consciousness for several weeks. A rupture of an aneurysm of anomalous right posterior communicating artery was found at autopsy.

in the hospital was progressively downhill. Death occurred on December 19, 1935, five days after onset.

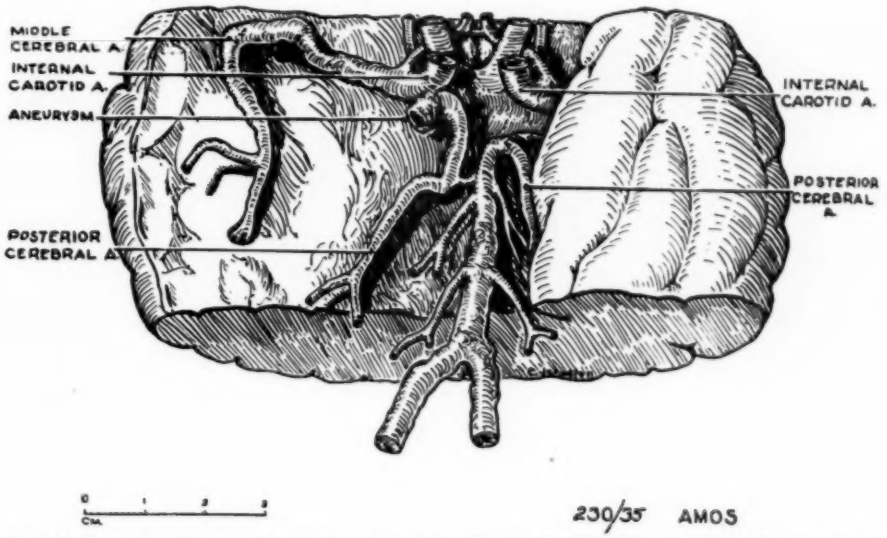

Fig. 6. Case vii. Pen and ink drawing of the base of the brain showing the point of rupture of the aneurysm arising from the anomalous right posterior communicating artery.

Clinical History. S. L. A., a colored female, seventy-two years of age, was admitted to the New Haven Hospital on December 16, 1935, in a semistuporous condition.

The patient had hypertension for at least twenty years with a systolic blood pressure at times as high as 250 mm. of mercury. In 1931 she had an attack similar to the present one, following which she was semistuporous for several weeks.

Two days before admission, the patient experienced a sudden severe sharp pain over the right eye. An hour later she vomited and that evening became semistuporous. The pain, although less severe, continued to the time of admission.

Physical examination showed the patient to be drowsy but rational. The blood pressure was 250 mm. of mercury systolic and 130 mm. diastolic. There was complete ptosis of the right eyelid. The right pupil was dilated and did not react to light. The left pupil reacted to light. The vessels of the fundus of the eye were sclerotic. The heart was enlarged to the left. The spinal fluid was bloody. The Wassermann reaction of the blood and spinal fluid was negative. On the following day, December 17, the extremities on the left side were flaccid and paralyzed and the reflexes were slightly more active than on the right side. The course

Necropsy. The postmortem examination was made by Dr. D. M. Grayzel. The heart weighed 610 gm. There was an ulceration measuring 0.5 cm. in diameter on one of the tricuspid valve leaflets. Several small verruccous nodules were present along the line of closure of the mitral valve. The aortic valve leaflets were thickened and contained several atheromatous plaques. The aorta was inelastic and was thickened by atheromatous plaques, some of which were calcified or ulcerated. The abdominal portion was slightly dilated and measured 7 cm. in circumference. The kidneys were not decreased in size, but contained numerous vascular scars.

Brain. There was a massive hemorrhage in the leptomeninges over the base of the brain and filling the entire ventricular system. The vessels of the circle of Willis were markedly thickened and sclerotic. The right posterior communicating artery arose in an anomalous fashion from the internal carotid artery and proceeded posteriorly as the posterior cerebral artery. From this latter vessel a small branch joined the basilar artery to complete the circle of Willis. At the anomalous junction of the internal carotid and posterior communicating arteries, which appeared like a side-to-side anastomosis of the two vessels, a small outpouching was present (Fig. 6). This had

ruptured to produce the subarachnoid and intraventricular hemorrhage.

COMMENT. Atherosclerosis and hypertension are important factors in the etiologic consideration of the aneurysm in this instance. To this, of course, must be added the anomalous condition of the right posterior communicating artery.

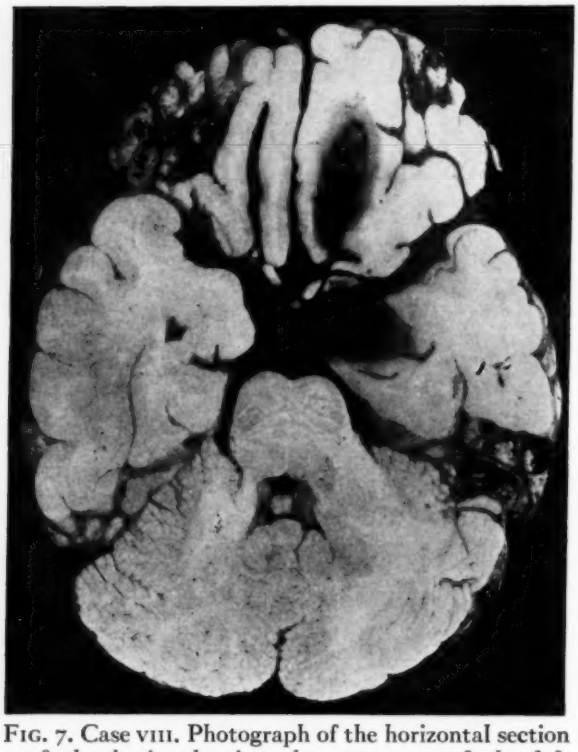

FIG. 7. Case VIII. Photograph of the horizontal section of the brain showing the aneurysm of the left internal carotid artery.

HYPERTENSIVE GROUP (WITHOUT ARTERIOSCLEROSIS)

CASE VIII. A white female of twenty-six years of age gave a history of scarlet fever five years previously. Her present illness was of sudden onset with nausea, severe headache and collapse, followed by convulsion, cervical rigidity and exaggerated deep reflexes, with a positive Kernig sign and markedly elevated systolic blood pressure. Death occurred nine days later. A rupture of an aneurysm of the left internal carotid artery and diffuse glomerulonephritis were the autopsy findings.

Clinical History. A. M. R., a white unmarried woman, twenty-six years of age, was admitted to the New Haven Hospital, August 22, 1918, because of headache and convulsions. At the age of five years she had had "spinal meningitis" and since then suffered from frequent headaches and "non-productive dry cough." She had diphtheria and scarlet fever at the age of twenty-one years. On the day before admission she was nauseated and had a severe headache, collapsing while on a trolley car. She vomited once. She had a convulsion, and was then brought to the hospital.

Physical examination showed irregular respirations resembling the Cheyne-Stokes type. The right pupil was larger than the left and both reacted sluggishly to light. The apex beat was most pronounced in the fifth interspace, 9.5 cm. from the midline. There was a long presystolic murmur heard best at the apex and the second sound was replaced by a soft murmur. There was no edema of the extremities. The systolic blood pressure was 230 mm. of mercury. Cervical rigidity was marked. The triceps, biceps, abdominal, patellar and Achilles reflexes were exaggerated but equal. There was a Babinski reaction on the left. Kernig's sign was marked on both sides, but more pronounced on the left.

Urinary excretion for the first twenty-four hours was 350 c.c.; the urine had a specific gravity of 1.012 and contained 0.12 per cent albumin, a few casts and a few white blood cells. The non-protein nitrogen of the blood was 79 mg. per 100 c.c. and the urea was 50 mg. per 100 c.c. A lumbar puncture yielded 60 c.c. of bloody spinal fluid. Smears and cultures of the spinal fluid were negative for organisms. The Wassermann reactions of the blood and spinal fluid were negative, and the colloidal gold reaction of the spinal fluid was 1112200000.

Bloody spinal fluid was obtained repeatedly during her stay in the hospital, which lasted nine days. Rigidity of the neck increased, but no further convulsions occurred. Signs of pulmonary congestion developed during the last two days. The systolic blood pressure on the day before death, August 31, was 130 mm. of mercury.

Necropsy. The postmortem examination was performed by Dr. F. P. McNamara whose findings are abstracted.

The heart was moderately increased in size, especially on the left, and was firm. There was no sign of endocarditis. The mitral valve was not stenotic. The lungs showed congestion of the lower lobes, and small patches of focal pneumonia and atelectasis. The kidneys to-

gether weighed 117 gm., were appreciably decreased in size and had granular surfaces. Microscopically they showed a chronic glomerulonephritis with considerable interstitial fibrosis and vascular thickening.

Brain. There was a large amount of clotted blood in the subarachnoid space over the surfaces of the brain. An aneurysm of the left internal carotid artery, 8 mm. in diameter, adjacent to the left optic nerve, seemed to be the source of the hemorrhage (Fig. 7).

A microscopic preparation through the aneurysmal dilatation showed a thin wall, but all the coats were easily distinguishable. At one point there was a proliferation of the subintimal connective tissue, and just beyond this the wall was ruptured. Attached to the ruptured wall and extending into the subarachnoid space was a fresh thrombus with no evidence of organization. The wall of a smaller vessel opening off the aneurysmal sac opposite the point of rupture appeared entirely normal.

COMMENT. Necropsy did not reveal any evidence of meningitis which the patient is said to have had at five years of age. This, then, can be excluded from consideration as regards the etiology of the aneurysm of the left internal carotid artery. It would seem in this instance that the hypertension which this patient had as the result of chronic glomerulonephritis following scarlet fever is the all important factor in the pathogenesis of the aneurysm. The site of the latter, namely, at the junction of the internal carotid artery and one of its branches, is a point of natural weakness in blood vessel structure. The combination of these two factors, the hypertension and the *locus minoris resistentiae*, must be employed to explain the formation and final rupture of the aneurysm in this case. The slight amount of subintimal connective tissue proliferation may possibly represent another factor in the formation of the aneurysm.

CASE IX. A white female of forty-three years had a history of hypertension and "neuralgic pain" on left side of face. The present illness had an onset with "neuralgic" attack, headache and loss of consciousness. There were bilateral positive Babinski sign and bloody spinal fluid. Death occurred in thirty-six hours. Autopsy showed rupture of aneurysm at the junction of the left internal carotid and anterior communicating arteries.

Clinical History. R. P., a forty-three year old white woman, was admitted to the New Haven Hospital August 17, 1919, in an unconscious condition. A year and a half before admission she was told that she had high blood pressure. For three years she had had attacks of "neuralgic pain" on the left side of the face, which she herself treated with proprietary medicines. On the day before admission, she had a severe "neuralgic" attack and on the following morning she felt weak and returned to bed complaining of headache on the right side. There were two brief periods of unconsciousness with apparent recovery in a short while. At 10:30 A.M. she became rigid, the muscles of the face twitched and she became unconscious.

Physical examination showed an obese, unconscious woman. Respirations were of the Cheyne-Stokes type. The right pupil was smaller than the left; both were constricted and neither reacted to light. The eye grounds showed engorgement of the veins, but no hemorrhages. The blood pressure was 96 mm. of mercury systolic and 62 mm. diastolic. The heart appeared enlarged to percussion, but the sounds were inaudible. The lungs were filled with moist bubbling rales, but no signs of consolidation were noted. The knee jerks could not be elicited; there was a positive Babinski reaction on either side. The urine contained a very slight trace of albumin, 2+ sugar, but no diacetic acid or acetone. The blood sugar was 490 mg. and the blood urea 17 mg. per 100 c.c. The spinal fluid was bloody. The Wassermann reaction of both spinal fluid and blood was negative.

The patient remained unconscious and died fifteen hours after admission to the hospital.

Necropsy. The postmortem examination was made by Dr. F. P. McNamara. The body weighed 145 lb. (66 kg.) and measured 61 inches (154 cm.) in length. The heart weighed 325 gm.; there was no hypertrophy but the right ventricle was dilated. The aorta showed a moderate degree of atherosclerosis. The lungs were edematous and there was passive congestion of the abdominal viscera. A small encapsulated mass of caseous necrosis, measuring 2 cm. in length and 0.5 cm. in diameter,

was seen in the liver. The esophagus was eroded and gastric contents had flowed through the perforation into the thoracic cavity, eroding portions of the pleura.

Brain. A massive subarachnoid hemorrhage covered the left hemisphere of the brain. At the junction of the left internal carotid with the anterior communicating artery the vessel was dilated to form an aneurysmal sac which was filled with clotted blood. This appeared to be the source of the hemorrhage.

Microscopically the aneurysm contained a recent thrombus. A portion of the wall was greatly thickened by hyalinized connective tissue, while one side was very thin, where the rupture had occurred. A section of the internal carotid artery very near the aneurysm showed a uniform thickening of the wall which encroached markedly on the lumen.

COMMENT. The two possible factors which played a role in the pathogenesis of the cerebral aneurysm are the hypertension and the natural weakness of the vessel wall at the site of the aneurysm. Evidence of hypertension exists only in the clinical record where it is stated that one and one-half years before her admission to the hospital she had high blood pressure. Anatomically, this evidence is only partially substantiated by the findings of atherosclerosis of the aorta and of the circle of Willis at the site of the vascular lesion. This evidence therefore, is merely suggestive and cannot be considered conclusive. As has already been noted in Case 11, the favorite site of a cerebral aneurysm is at the junction of vessels forming the circle of Willis where a *locus minoris resistentiae* presumably exists. In this instance the marked thickening by hyalinized connective tissue of a part of the aneurysmal wall may have been an important factor both in the formation of the aneurysm and its subsequent rupture.

SYPHILITIC GROUP

CASE X. A white male of forty-nine years presented symptoms of general paresis. He had a right hemiplegia and loss of speech five years ago followed with partial recovery but persisting speech defect and irritability, ataxia and weakness of the left leg. There was a sudden onset of paralysis of the left arm and leg after a fall and loss of consciousness followed by Jacksonian convulsions. Death from bronchopneumonia and cardiac decompensation occurred twelve days later. On autopsy, meningovascular syphilis with multiple syphilitic aneurysms of the right middle cerebral artery was found.

Clinical History. J. S., a white man of forty-nine years, was admitted to the New Haven Hospital March 10, 1930, in a comatose condition.

Previous to 1924 mental changes had been noted consisting chiefly of grandiose ideas. In December, 1924, he developed right hemiplegia and loss of speech. Locomotion gradually returned but his speech remained thick and the patient remained irritable. After December, 1929, he had considerable difficulty in walking, and on several occasions his left leg gave way under him, causing him to fall. Since March 5, 1930, he had had twitching spasms on the right side associated with loss of consciousness. On the day of admission, he fell and the left arm and leg became paralyzed.

Physical examination showed paralysis of the left arm and leg and left facial weakness. The retinal vessels were sclerotic and there was a small hemorrhage in the left optic disc. The heart was enlarged to the left; the blood pressure was 210 mm. of mercury systolic and 120 mm. diastolic. The reflexes were hyperactive, particularly on the left, and there was a positive Babinski reaction on the left. The Kahn test of the blood was 4 plus.

The patient gradually regained consciousness. On March 13 he had a Jacksonian convulsion without loss of consciousness. His general condition improved for a time, but evidences of pneumonia, consolidation and cardiac decompensation appeared and he died on March 17, 1930, twelve days after the onset of the final episode.

Necropsy. The postmortem examination was made by Dr. S. W. Stringer. The heart weighed 550 gm.; there was marked hypertrophy of the left ventricle and dilatation of the right ventricle. The aortic valve was competent. The aorta showed numerous elevated and ulcerated atheromatous plaques, and a few puckered linear and stellate scars. The media was free of cellular infiltration on microscopic examination. The lungs were congested and

contained small patches of pneumonia. The kidneys were of the congenital polycystic type.

Brain. The large vessels at the base of the brain were greatly thickened and inelastic. This was especially true of the right middle cerebral artery which showed three distinct aneurysmal dilatations along its course (Fig. 8). The largest of these, measuring 6 to 7 mm. in diameter, was situated at the bifurcation of the vessel into its three main branches. The smaller aneurysms, 2 to 3 mm. in diameter, had no relationship to the branching of the artery.

Microscopically, the right middle cerebral artery at the site of the aneurysm was greatly thickened by atheromatous change in which cholesterin crystal spaces and calcium salt deposition in the intima were prominent. The abundant subintimal connective tissue was hyalinized. The smooth muscle of the media was almost completely replaced by hyaline connective tissue, and only a few small fragments of elastic tissue were demonstrable. Infiltrations of lymphocytes occurred frequently in the media and adventitia. At one point in the thickened intima there was an endarteritis characterized by mononuclear cell infiltration. Superimposed on this was a thrombus which filled about one-third of the lumen of the artery. The opposite wall of the artery, where the dilatation occurred, was very thin and was composed of hyalinized connective tissue.

The leptomeninges over the brain were thickened and milky. They contained an exudate of lymphocytes and plasma cells which was concentrated around pial vessels. Numerous areas of anemic and hemorrhagic infarction, which appeared to be of different ages, were present in the right cerebral hemisphere. There were also smaller areas, especially in the right internal capsule, which resembled small gummata, but no spirochetes were demonstrated in Jahnel preparations. The midbrain and pons showed pyramidal tract degeneration.

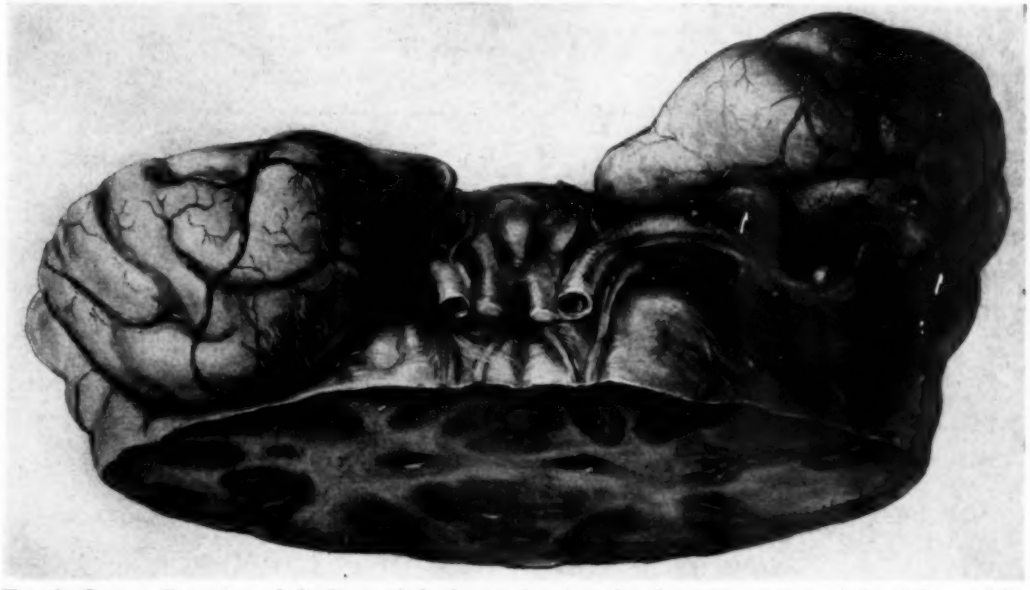

Fig. 8. Case x. Drawing of the base of the brain showing the three aneurysms of the right middle cerebral artery.

COMMENT. Syphilitic arteritis is the underlying factor in the formation of the three aneurysms found in the right middle cerebral artery. As is often the case in vascular syphilis, superimposed atheromatous changes complicate the infectious lesion. The multiplicity of aneurysms is worthy of note as is also the fact that in spite of the marked hypertension none of the aneurysms ruptured.

Case xi. A colored female of forty-nine years with a long history of syphilis, hypertension and cardiac enlargement, died from intractable cardiac decompensation. Upon autopsy meningovascular syphilis and aneurysms of both internal carotid arteries with compression of left oculomotor nerve were demonstrated.

Clinical History. J. B., a colored female of forty-nine years, was admitted to the New Haven Hospital on November 2, 1934, because of shortness of breath and abdominal pain.

FIG. 9. Case XI. Drawing of the base of the brain to show the two saccular aneurysms on the internal carotid arteries. The inset shows the laminated blood clot which partially filled one of the aneurysms.

The patient was in a hospital in 1928 with a complaint of being "run down" and was told she had syphilis. In 1931 she developed a left facial paralysis. At that time her blood pressure was 220 mm. of mercury systolic and 140 mm. diastolic. The heart was enlarged. The pupils were irregular and fixed to light. Roentgenograms showed a generalized dilatation of the ascending and transverse aorta. The Kahn reaction of the blood was 4 plus. In December, 1932, she complained of a severe headache and a sense of fullness in the head; this gradually disappeared. She was in this hospital from July 16, 1935, to October 25, 1935, because of cardiac decompensation which improved under treatment. The cardiac decompensation returned quickly after she was discharged and she was readmitted November 2, 1935.

Physical examination showed an emaciated colored woman who was quite dyspneic and orthopneic. Other findings were essentially the same as had been recorded in 1931. The Kahn test of the blood was 4 plus, the Wassermann reaction of the spinal fluid was 4 plus and the colloidal gold curve of the spinal fluid was 5555544311.

In spite of careful and rigorous treatment, the cardiac decompensation increased, and the patient died on February 13, 1935. At no time had there been any evidence of oculomotor disturbance.

Necropsy. The postmortem examination was made by Dr. D. S. Egbert. There was extensive edema of the subcutaneous tissue, but little fluid in the body cavities. The heart weighed 700 gm.; both ventricles were hypertrophied; softened mural thrombi were present in the right auricle and left ventricle; the commissures of the aortic valve leaflets were slightly separated but the leaflets themselves were thin and pliable. A fairly well circumscribed dilatation of the aorta was present just proximal to the innominate artery. This contained atheromatous plaques and a few stellate

depressed scars. The descending portion of the thoracic aorta likewise contained numerous sclerotic plaques and stellate scars. Microscopically, the aorta showed scarring of the media and fairly prominent perivascular infiltrations of lymphocytes. The arrangement of the elastic fibers was distorted. The intima showed typical atheromatous changes.

Brain. There was a large thrombosed aneurysm of the left internal carotid artery which compressed the lateral aspect of the optic chiasm and also the left oculomotor nerve (Fig. 9). This aneurysm was situated just behind the left posterior communicating artery. There was also an aneurysm of the right internal carotid artery which was in the same position and of the same size as the one on the left, but it was not thrombosed, and it did not cause compression of the optic chiasm or right oculomotor nerve. Microscopically, the thrombus in the left internal carotid artery almost occluded the lumen and showed beginning organization at its periphery. The wall of the vessel was thin and hyalinized, and the smooth muscle of the media was almost completely replaced by fibrous tissue. The media was vascularized and contained numerous lymphocytes and plasma cells and phagocytes laden with ingested blood pigment.

The pia-arachnoid was thickened and milky in appearance, and contained numerous lymphocytes and plasma celll. The dorsal columns of the spinal cord showed demyelinization and gliosis. A large, liquified anemic infarct was present in the left occipital lobe involving the ventral limb of the calcarine cortex.

COMMENT. Syphilitic arteritis is quite plainly the underlying factor in the formation of the aneurysm of each of the internal carotid arteries. The clinical history of syphilis, the serological and spinal fluid findings and the microscopic appearance of the aneurysms all support this diagnosis. Again this patient has a marked hypertension which may be of importance in connection with the aneurysmal formation.

CONGENITAL GROUP

CASE XII. A white male of two years sustained a fractured skull in an automobile accident, with apparent recovery in five days. Onset of irritability two days later was followed by convulsion, dyspnea and cyanosis. Repeated ventricular puncture was done to relieve intracranial pressure. There was a sudden flow of arterial blood during a ventricular puncture. Death ensued thirty-six days after the accident and twenty-nine days after the onset of subsequent symptoms. A rupture of an aneurysm of the right vertebral artery was found at autopsy.

Clinical History. A. F., a white male of two years and nine months, was admitted to the New Haven Hospital October 14, 1929, because of restlessness, cyanosis and convulsions.

On September 22, 1929, the patient was thrown from an automobile which overturned. He was unconscious for a time but cried a little. A roentgenogram showed two small fractures of the left frontal bone. After five days in the hospital he appeared entirely well and was discharged. Two days later he became irritable and cried for long periods. Three days after this there was a convulsion, followed by cyanosis and dyspnea. The temperature varied from 101°F. to 105°F. for about a week. Another convulsion occurred, followed by restlessness and insomnia, and he was admitted to this hospital.

Physical examination showed a comatose child having marked respiratory difficulty and occasional spasms of the arms and legs. The neck was retracted and rigid. The respirations were of the Cheyne-Stokes type. The spinal fluid was bloody and the initial pressure was 300 mm. of water.

Repeated lumbar punctures yielded blood tinged or frankly bloody fluid under increased pressure which on one occasion was over 700 mm. of water. Ventricular puncture was made on October 24, at which time 210 c.c. of bloody fluid were removed. This procedure was repeated three times. On the last occasion, after the removal of 70 c.c. of xanthochromic fluid, there was a sudden spurt of arterial blood. The blood pressure rose immediately to 156 mm. systolic and 86 mm. diastolic, and the pulse rate to 160. Two more ventricular punctures were made to relieve the increased pressure, but without effect. The respirations became irregular and ceased; death occurred on October 24, 1929, thirty-six days after the automobile accident and twenty-nine days after the onset of subsequent symptoms.

Necropsy. The postmortem examination was made by Dr. H. M. Zimmerman. The head

was large and hydrocephalic. The suture lines of the skull were separated.

Brain. The brain was large and soft and

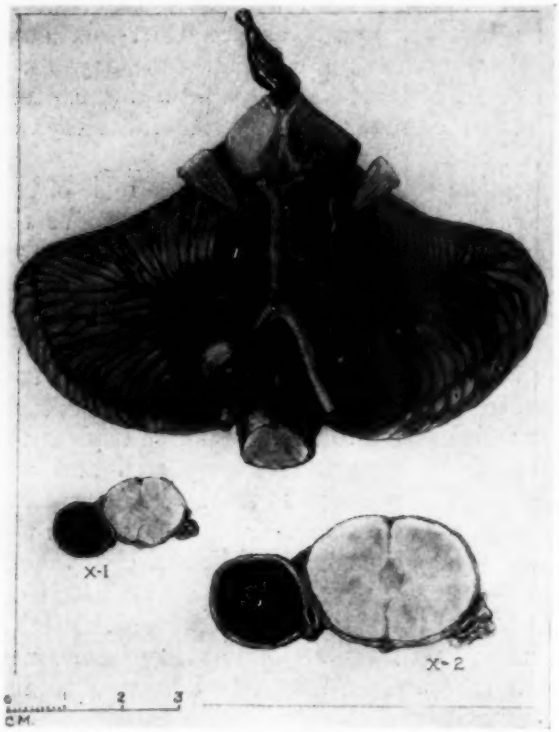

FIG. 10. Case XII. Drawing to show the aneurysm on the right vertebral artery. The inset shows the thrombus filling the aneurysm.

was covered by a massive subarachnoid hemorrhage over all its surfaces. The ventricular system was markedly dilated and filled with clotted blood. On the lateral surface of the right vertebral artery, just before its junction with the left to form the basilar artery, there was a firm nodule which measured 1 cm. in diameter (Fig. 10). On section this was seen to be an aneurysmal sac filled with a laminated blood clot. Microscopically, the subarachnoid hemorrhage showed complete laking of the blood. The aneurysmal sac was seen to be definitely an outpouching of the lateral wall of the right vertebral artery. For about three-fourths of its circumference the wall of this vessel was well formed and normal; then quite suddenly it became thin and was evaginated to form the aneurysm. The media was narrow and the elastic lamella appeared to be malformed. Half the aneurysm adjacent to the artery was filled with a laminated blood clot, while the remainder was filled with a mass of red blood cells without definite arrangement. The outer portion of the wall was fragmented and showed evidence of recent rupture by the presence of large masses of preserved red blood cells in the subarachnoid space surrounding the aneurysm.

COMMENT. A fracture of the skull during an automobile accident is a prominent feature in the clinical record of this patient. The onset of cerebral symptoms following this accident was after a period of only seven days. The unusual site of the aneurysm in the right vertebral artery at a point not related to a bifurcation is further evidence in support of trauma as the important etiologic factor in its formation. In spite of all this the youth of the patient, two years, and the fact that the microscopic appearance of the aneurysm suggests a malformation of the vessel wall are factors pointing to a congenital basis for the vascular lesion. The question as to etiology on a traumatic or congenital basis cannot be decided absolutely.

UNDETERMINED

CASE XIII. A white male of forty years collapsed suddenly and died almost immediately. A rupture of an aneurysm of the lateral branch of the left posterior inferior cerebellar artery was discovered at autopsy.

Clinical History. C. H. F., a white male of forty years suddenly collapsed while talking to a business acquaintance and died almost immediately. He had not complained of any symptoms during the day of his death; information regarding his previous history was not available.

Necropsy. The postmortem examination was made by Dr. M. T. Shelton. The heart weighed 350 gm. and showed no abnormalities. There were very slight atheromatous changes in the aorta. The kidneys together weighed 250 gm. and were very slightly granular.

Brain. The subarachnoid space over the base of the brain and over both temporal and parietal lobes was filled with a massive fresh hemorrhage which also filled the entire ventricular system. The blood vessels at the base of the brain contained a few small atheromatous plaques but this process was not extensive. The left lateral branch of the posterior inferior cerebellar artery just before it enters the choroid plexus contained a small aneurysm

which measured 3 or 4 mm. in diameter (Fig. 11). This aneurysm appeared to be the source of the subarachnoid and intraventricular hemorrhage. Microscopically, the intima of the artery and the aneurysm were normal in appearance. The media, however, was almost acellular, hyalinized and fairly thick except near the point of rupture where it became quite thin. There was no calcification or atheromatous change in the vessel wall and no cellular reaction. The adventitia was filled with fresh blood and also contained a small amount of old blood pigment.

COMMENT. An insurmountable handicap is encountered in the attempts to evaluate the pathogenesis of the aneurysm in this instance. This is due to the entire lack of a preceding clinical history as well as to an almost complete absence of vascular disease. It is unjustifiable to assume that this patient had hypertension, but such a possibility cannot be denied. The position of the aneurysm is rather unusual and aids but little in the interpretation of its etiology. Aside from the possibility of hypertension as its underlying basis, congenital malformation of the lateral branch of the posterior inferior cerebellar artery has to be considered.

DISCUSSION

In this series of cases, only 2 of the 13 intracranial aneurysms produced sudden death (cases XIII and IV). In the first instance death was immediate and in the case of the second patient, death occurred a few hours after the sudden onset of symptoms. The remaining patients with ruptured aneurysms lived for varying periods of one to twenty-nine days after the initial symptoms of rupture. In the first instance of sudden death in the series (case XIII) no past history was obtained. In the other case, however, there had been severe headaches for several years. The occurrence of headache, migraine or "neuralgic pain" is fairly common in intracranial aneurysms, 6 of our patients having had a definite long standing history of such symptoms. A symptom of this type is quite prominent in retrospect, but clinically is usually of such indefinite character that its value in making an antemortem diagnosis is probably only of questionable value.

FIG. 11. Case XIII. Drawing to show the aneurysm of the lateral branch of the left posterior inferior cerebellar artery.

In 7 patients of our series the onset of symptoms due to rupture of the aneurysm was quite sudden. In another patient with sudden onset of symptoms (case V) the aneurysm did not rupture, and the symptoms were due to thrombosis of the aneurysm with extensive cerebral infarction. Three cases (II, IX and XII) had gradual onsets which, however, progressed in the course of a few hours (cases II and IX) or days (case XII) to advanced signs of intracranial damage.

Four cases in this series (cases II, III, VI, XII) showed definite evidences of repeated hemorrhages from the aneurysms. One additional case (VII) had a history of an attack five years previously which was said to have been similar to the one which led to the death of the patient. Cushing has pointed out the frequency with which such recurrent attacks of "weeping" from

intracranial aneurysms occur, and the importance of this evidence in making a proper clinical diagnosis.

The onset of symptoms with sudden stabbing pain over one eye occurred in 4 of our cases (cases II, III, VII and IX). This symptom, although it is present in a rather small number of cases, is so striking when it occurs that it should be a definite aid not only in making a diagnosis of ruptured aneurysm but also in localizing the site of rupture. In case IX there was a long history of "neuralgic pain" on the left side of the face and the terminal illness was ushered in with a severe attack of neuralgic pain. This may perhaps be analagous to the pain over the eye experienced by the 3 other patients, though it is not typical.

The 3 patients with typical sudden stabbing pain over the eye all developed paralysis of the oculomotor nerve on that side (cases II, III, VII). These were the only patients in the series who developed such paralysis. The necropsy on one other patient (case XI) showed definite compression of the oculomotor nerve by the aneurysm but there had not been clinical evidence of oculomotor paralysis, and no pain over the eye had been noted.

Wagener and Foster recently have reported 2 cases of ruptured intracranial aneurysm associated with hemorrhages into the retina. In our series of cases, 3 patients showed retinal hemorrhages (cases I, IV, X). In none of these was there oculomotor paralysis. The 2 cases reported by Wagener and Foster likewise did not have palsy of the oculomotor nerve. It seems, therefore, that these two symptoms, although occurring frequently, are seldom present in the same individual.

Of the 13 cases in this series, only 2 were of syphilic origin (cases X, XI). There is considerable disagreement in the literature concerning the importance of syphilis as a factor in the production of intracranial aneurysms. More recently, however, writers on the subject state that syphilitic aneurysms of the cerebral vessels are much less common than had been considered previously, and constitute only a small percentage of all intracranial aneurysms. It is interesting to note in this series that the only instances of multiple aneurysms occurred in the 2 syphilitic cases.

Another interesting finding is that in only 3 cases did the aneurysms fail to rupture and both syphilitic cases were in this group. The third unruptured aneurysm was of arteriosclerotic origin (case V). The relationship of etiology to the multiplicity of aneurysms and to their tendency to rupture is difficult to evaluate, but it appears from this series that syphilitic aneurysms are frequently multiple and are less prone to rupture.

The spinal fluid was not examined clinically in 3 of the patients in our series (cases I, IV, XIII). In 3 cases (V, X, XI) the spinal fluid was free of blood. These were the 3 patients in whom the aneurysms did not rupture. All the remaining 7 showed bloody spinal fluid. At necropsy, 7 of the patients revealed massive intraventricular hemorrhage as well as subarachnoid hemorrhages (cases I, III, IV, VI, VII, XII, XIII). It is to be noted that this group includes the 3 instances in which the spinal fluid was not examined clinically. It is evident, therefore, that blood in the spinal fluid is the most frequent finding in cases of ruptured intracranial aneurysm.

The necropsies in 2 of our cases (VI, IX) showed erosion of the lower portion of the esophagus with the escape of gastric contents into the pleural cavities. In case VI there were hemorrhages in the tuberal and other nuclei of the hypothalamus, which were associated with the intraventricular hemorrhage. Recent work by Hoff and Sheehan tends to indicate a causal relationship between lesions of the tuberal nuclei and gastric or esophageal erosions. The work has been chiefly experimental and as yet comparatively few clinical reports have appeared. Case IX was seen in this hospital in 1919 and the material is not available now to enable a study of the hypothalmic region.

Two of our cases (v, vii) showed anomalies of the circle of Willis associated with aneurysm. In both instances the vessels were markedly arteriosclerotic, and it is difficult to evaluate the importance of the anomalies as a causative factor in the formation of the aneurysms.

CONCLUSIONS

The histories and necropsy findings of 13 cases of intracranial aneurysm are presented in detail. From their study the following conclusions can be drawn:

Rupture of intracranial aneurysms must be considered in cases of sudden death. In a majority of instances there will be clinical signs and symptoms which may lead one to suspect that a cerebral aneurysm has ruptured. Some instances occur in which the clinical findings are so striking that the correct diagnosis should be evident.

From a medicolegal aspect, as well as from the point of view of general pathology, the important consideration is the evaluation of the factors which lead to the formation and rupture of an aneurysm. In many instances this is extremely difficult or wholly impossible. Arteriosclerosis undoubtedly plays the major role in a large number of cases, particularly in the older age group. Syphilis, on the other hand, is probably rather unimportant as an etiologic factor. The role which congenital anomalies of vessels, congenital aneurysms, trauma, and hypertensive states due to causes other than arteriosclerosis play in the formation and rupture of aneurysms is difficult to evaluate.

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RUPTURES AND TEARS OF MUSCLES AND TENDONS*

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RUPTURES and tears of muscles and tendons have been considered rare lesions until recently. Many cases, however, have been overlooked on account of incomplete physical examinations, and too often it has been the tendency for the average clinician to mistake muscle and tendon ruptures and tears for sprains, bursitis and arthritis. There are certain signs and symptoms which are characteristic of each ruptured tendon or muscle.

Early diagnosis in these cases prevents unnecessarily prolonged treatment and in many instances a permanent disability. While it is true that we should rely mainly on physical examinations in making a diagnosis of a ruptured or torn muscle or tendon, we have been aided in certain instances by using a radiographic technique which brings out the soft structures. This procedure, of course, is very limited and can only be applied to the larger tendons and muscles.

ETIOLOGY

Ruptures and tears of muscles and tendons may be seen in any age group. Involvement of almost all the important muscles and tendons have been reported. They may result from direct or indirect trauma in any case, but there are certain predisposing factors which are of importance and Gilcreest[1] has presented these in outline form which are as follow:—

1. Senility
2. Pathologic changes
   (a) Arthritis
   (b) Myositis
   (c) Acute infectious disease
   (d) Arteriosclerosis
   (e) Syphilis
   (f) Tuberculosis
   (g) Neoplasm
3. Physiologic predisposition
4. Occupation
5. Fatigue
6. Trauma
   (a) Indirect
   (b) Direct

The changes which occur with senility naturally predispose to rupture of tendons and muscles; therefore any acute or chronic, local or systemic disease, plays an important role in predisposition. Occupation is important because certain laborers are more likely to sustain ruptures of certain tendons than others, due to the heavy strain put upon the muscles in addition to other predisposing factors. It is a fact that these people are more subject to direct trauma than others of less hazardous occupations. Gilcreest[2] states that excessive fatigue may render muscular fibers less likely to resist a sudden and forceful pull or jerk, and that fatigue superimposed upon other predisposing factors may cause rupture of a tendon to be produced much easier.

MECHANISM

In considering the rupture of muscles and tendons it is important to recognize the

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mechanism involved in producing these injuries. Brickner and Milch[4] presented in outline form conditions that may lead to muscle ruptures, which was based upon the observations of Madyl. These are as follows:—Muscles tear as a result of

I. Muscular Action
  A. Flaccid
  (1) Contraction of antagonist
  (2) Passive stretching (no case ever reported but theoretically possible)
  B. Contracted
  (1) Active contraction
  (2) Contraction of antagonist
  (3) Increase of tearing over cohesive force
  (4) Asynchronic contraction
    (a) Inner part against outer (as in bifid muscles)
    (b) Distal part against proximal part
  (5) Additional muscular force of another muscle
II. External trauma
III. Spiral twists of limb
IV. Degeneration of muscle as in neurological conditions, disease, etc.

SITES OF MUSCLE AND TENDON RUPTURES

Statistics on ruptures and tears of muscles and tendons usually list the following ruptures in the order of their frequency: muscles of the calf; extensors of the leg; biceps, Achilles tendon and extensor of the thumb; and less frequently involved are the supraspinatus, rectus abdominis, extensors of the fingers and thumb, adductors of the thigh and triceps brachii. If all the so-called sprains and bruises and other injuries could be exposed at operation it is possible that many other muscles would be found to be the site of either partial or complete rupture.

RUPTURES OF THE BICEPS BRACHII

The biceps muscle may undergo rupture at almost any site. Gilcreest,[1,2] in a recent and most elaborate discussion of the subject, states that Bianchieri has reported that the long head is involved in 96 per cent of the cases, the lower tendon in 3 per cent and the short head in 1 per cent. The reader is referred to the article of Gilcreest for more detailed consideration of rupture of the long head of the biceps.

Because of its passage through the bicipital groove and the shoulder joint the causative factors involved in ruptures of the long head are different from any other tendon. Due to disease processes, such as arthritis, in the shoulder joint and the fraying out of the tendon from constant irritation of the tendon passing through the bicipital groove over a long period of time, the tendon weakens and is much more subject to rupture when a strong force is applied. The actual force that causes the final rupture is usually a sudden forceful contraction of the muscle.

SYMPTOMS AND SIGNS

The symptoms of a rupture of the long head of the biceps are variable. Usually there is a history of exertion or sudden strong contraction of the biceps with a sharp pain in the shoulder and a snap or crack which can sometimes be heard several feet. If the rupture occurs in an already diseased tendon, however, there may be very little pain at the time and the only symptom is weakness of the arm and a vague feeling of discomfort in the shoulder. If the rupture occurs in the long head, the belly of the muscle will be prolapsed downward in contraction. If it occurs in the lower tendon, which is rare, the entire muscle will retract upward when contracted.

Huerter's sign, flexion of the forearm in pronation when the biceps are tense is more forcible than when the forearm is supinated and the biceps relaxed, is often helpful in the diagnosis.

Ludington's sign[5] which is also important is elicited in the following manner:—The patient is directed to rest his folded hands, palms down on the top of his head and allow the interlocked fingers to support the weight of the arms. In this position there is maximum relaxation of the long head. The

examiner then places two fingers on the tendon of the long head of the biceps in each arm, and directs the patient to simultaneously contract and relax his biceps muscles. The contraction of the long head tendon on the sound side is plainly felt while it is absent on the affected side if the tendon is ruptured.

Yergason's sign is of aid in diagnosing wear and tear of the long head of the biceps and synovitis of its tendon sheath. Yergason describes it as follows: "If the elbow is flexed to ninety degrees, the forearm being pronated, and the examining surgeon holds the patient's wrist so as to resist supination and then directs that active supination be made against his resistance, pain, very definitely localized in the bicipital groove indicates a condition of wear and tear of the long head of the biceps, or synovitis of its tendon-sheath."[6]

TREATMENT

Partial ruptures and tears of the biceps may be treated conservatively by immobilization for two or three weeks. Complete ruptures should be operated upon in all instances if the general condition of the patient permits. In ruptures of the long head it is not necessary to open the shoulder joint in an effort to replace the tendon into its original site and this procedure does not seem justified in view of the excellent results obtained by other procedures. Gilcreest[2] states that the long head may be sutured to one of the following: (1) the coracoid process; (2) the tendon of the short head of the biceps; (3) the pectoralis major; (4) the sulcus inter-tubercularis; (5) the deltoid. Gilcreest further states that he uses a combination of two of these methods, i.e., suture of the long head through the short head and also suture of the long head to the coracoid process, thus giving a double attachment. We also favor this procedure. When ruptures occur in the middle of the tendon or in the muscle proper, repair should be done as in any other muscle or tendon rupture. Ruptures of the lower tendon usually take place at the junction of the tendon and bone and for this reason it is necessary to reattach the tendon to the radius.

REPORT OF CASES

Case I. P. R., Negro, male, aged thirty-eight years, was admitted to the Orthopaedic Service of the T. C. I. Employees' Hospital, December 2, 1927. He stated that shortly before admission while holding a drill handle (the position of the forearm being forcible flexion and supination) his foot slipped and an ore car ran over his right leg, suddenly pulling his arm and hand from his hold on the drill handle.

Examination on admission to the hospital (Fig. 1) revealed only slight abrasion of the skin but a definite subcutaneous rupture of the main belly of the right biceps flexor cubiti, a laceration over the right frontal region and a severe compound communited fracture of the middle third of the right tibia and fibula. Operation was performed immediately under general anesthesia. A linear incision was made over the site of rupture and the muscle exposed, revealing a complete rupture of both bellies of the biceps in the middle third. The edges of the muscle were smooth and gave the appearance of having been cut by a knife. The torn edges of the muscle were brought together and sutured with chromic catgut, good approximation being obtained. A posterior plaster molded splint was applied to the arm in acute flexion. The compound fracture of the right leg was also properly set at this time. The wound healed without infection and the patient regained full function of the arm.

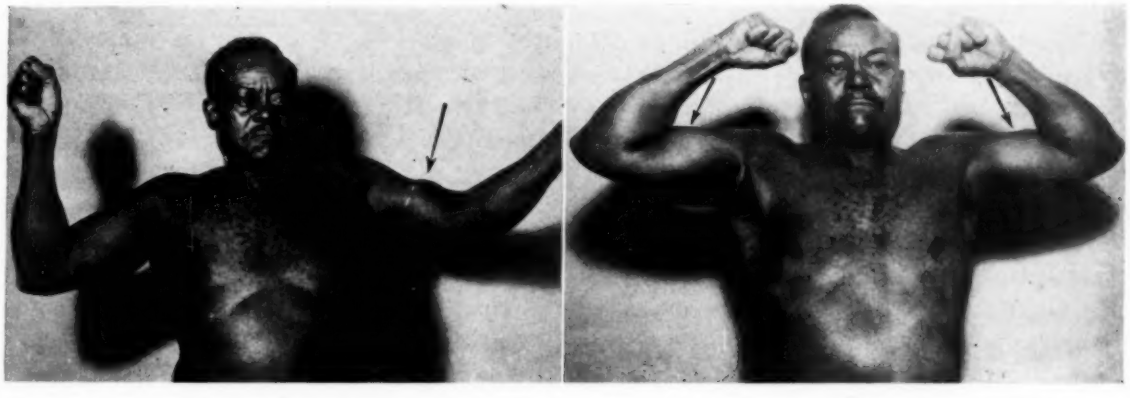
Case II. R. F. B., white male, aged sixty-four years, was admitted to the Employees' Hospital, September 26, 1929, one week after having received an injury to the right shoulder and arm. He stated that while shoveling, when he lifted a shovelful he noticed a snap in his right shoulder accompanied by a sudden severe pain and weakness in the arm. He quit work immediately and went home where he remained in bed applying heat to the shoulder until he was admitted to the hospital. A diagnosis of a rupture of the muscular part of the long head of the right biceps (Fig. 2) was made and operation under local anesthetic was performed the next day. An incision made over the site of rupture revealed a rupture of the muscular portion of the long head of the biceps. The muscle ends were united by chromic catgut

sutures. A posterior molded plaster splint was applied with the arm in acute flexion at the elbow.

to the edge of the glenoid. There were no evidences of associated joint disease. The long tendon was fixed to the coracoid process with

Fig. 1. Case I. Showing a rupture of the right biceps muscle. (Conwell, H. Earle. *Jour. Bone and Joint Surg.*, 10: 788–790 [Oct.] 1928.)

Fig. 2. Case II. Showing a typical deformity following a rupture of the long head of the biceps.



A B

Fig. 3. Case III. A. Shows the deformity produced by a rupture of the long head biceps; B. the end results after operation. There is full function of the left upper extremity.

Recovery was uneventful and the patient soon regained full function of the extremity.

Case III. W. G., a colored male, aged fifty-five years, was admitted to the Employees' Hospital, December 1, 1933. He stated that one week previously when he was doing some heavy lifting he picked up a load in undue haste when he heard and felt something snap in his left shoulder. There was also a sudden severe pain in the shoulder, with loss of strength in the arm. Examination on admission revealed typical signs and symptoms of a rupture of the tendon of the long head of the biceps (Fig. 3A). The patient was otherwise in good physical condition. Operation was performed through an incision made high upon the arm along the tendon of the long head. Exposure of the tendon revealed a rupture near its attachment chromic catgut. The arm was immobilized in flexion for three weeks when gradual active motion was begun. Recovery was practically complete in four months. There was complete return of function after a year and the patient stated that the injured arm was better than the other (Fig. 3B).

RUPTURE OF THE QUADRICEPS MUSCLES AND TENDON

Rupture may take place in almost any of the muscles or tendons of the thigh; that of the adductor muscles is not infrequent and is called "rider's muscle" because it occurs so often in horseback riders. The quadriceps tendon may rupture at its attachment to the patella or at any point

above it, and may be caused by direct or indirect violence. A direct blow on the quadriceps tendon may result in a complete rupture, as occurred in one patient who was kicked above the knee by a mule. The most common cause of a rupture of this tendon, however, is by muscular action or overstretching of the quadriceps. This is best illustrated by an individual who is stepping down a step and the knee flexes under the body weight, the individual suddenly contracts the quadriceps in an effort to keep from falling and the quadriceps tendon gives way under the sudden powerful contraction of the muscle. Many cases have been reported as caused by overstretching, as when an individual falls in a sitting position on the flexed leg forcing flexion beyond the normal degree. These are also the causes of fractures of the patella and Sonnenschein[7] points out that due to some preexisting disease the quadriceps is the first to break. Rare cases of bilateral rupture of the quadriceps have been reported.

The diagnosis offers no problem in complete ruptures but many cases have been overlooked for a long time, being treated for sprains or other conditions. There is a history of injury, with complete or partial loss of extension of the leg with swelling and pain in the region of the knee. The patient may be able to extend the leg partially on the thigh but this is because a few fibers are left intact and allow some function. A definite gap can be felt in the quadriceps tendon above the patella which clinches the diagnosis. Lateral roentgenograms often show a defect in the tendon or a detached particle of the patella.

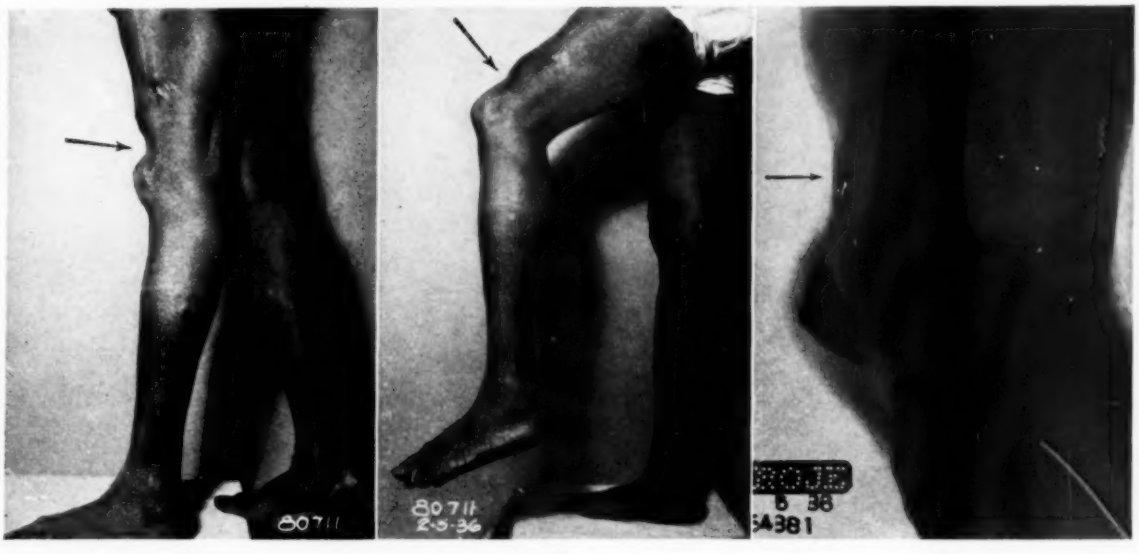
It is important that the diagnosis be made early so that proper treatment can be instituted. If this is not done the quadriceps undergoes early atrophy, fibrosis and retraction so that later operation is a formidable procedure. Partial ruptures may be treated conservatively by immobilization in extension, in a plaster cast for two or three weeks if the rupture is not too extensive. Complete rupture, however, demands immediate surgical repair if full functional results are to be obtained.

Operation should be performed under a general anesthetic if possible in order to obtain relaxation of the muscles so that reapposition of the ends of the tendon will be facilitated. The ends of the tendon should be exposed either by a longitudinal or a horseshoe shaped incision over the patella with the convexity downward. The ends of the tendon should be smoothed and approximated and sutured substantially with either chromic catgut, kangaroo tendon or living fascia. The lateral expansions of the quadriceps should also be sutured as usually they are torn quite extensively. If the tendon is separated from the patella it will be necessary to fix the tendon to the patella by drill holes in the upper end of the bone. The leg should be immobilized in full extension in a plaster cast for about four weeks, when gradual active motion, massage and physiotherapy should be begun. Active motion earlier than this is not advisable because the tendons heal so slowly that separation at the suture line would be a danger. The prognosis is good and full functional results are usually obtained. A few cases have been reported, however, where the tendon ruptured the second time and another operation was subsequently necessary.

REPORT OF CASES

Case IV. D. R., colored male, aged sixty-four years, was admitted to the Employees' Hospital, October 21, 1933. He stated that three weeks previously his left foot slipped when he stepped on a small rock while walking down a hill and he fell with the left foot and leg flexed under his buttocks. He felt immediate pain in the region of the left knee and was unable to extend the leg. He consulted a physician a few days later who advised him to come to the hospital at once. The patient finally consented to be hospitalized because he noticed that he could not go up and down steps as usual. Examination upon admission showed moderate swelling in the region of the left knee with inability to extend the leg on the thigh. The patella was abnormally movable from side to

side and there was a large defect, the width of three fingers in the quadriceps tendon above the patella. The patient also showed evidence revealed a retraction upward of the torn quadriceps tendon with a small chip off the patella (Fig. 5C).



A B C

FIG. 4. Case IV. A. Shows old deformity resulting from a complete rupture of the quadriceps tendon which was not operated; B. showing attempt at extension of the leg; very little extension was possible; C. roentgenogram showing marked retraction of the quadriceps tendon.

of senility and arteriosclerosis with hypertension. A diagnosis of rupture of the quadriceps muscle was made and operation advised but was refused. The knee joint was aspirated with removal of 35 c.c. bloody fluid. A circular plaster cast was applied to the foot and leg with the leg in full extension. The cast was removed in four weeks, after which time there was no evidence of union of the tendon ends. The patient was not seen again until requested to report to the clinic for observation in February, 1936, over two years after the injury. At that time there was a large visible defect above the patella (Fig. 4A), and the patient could only slightly extend the leg (Fig. 4B). He suffers much inconvenience from the injury chiefly because he could not walk up or down a hill or steps unless he advanced sideways with the use of a cane. He still refuses operation.

CASE V. H. F., colored male, aged forty-five years, was kicked above the knee by a mule and was admitted the same day to the Employees' Hospital, August 7, 1933. Examination revealed swelling of the knee joint with pain and inability to extend the leg. There was considerable lateral motion of the joint indicating rupture of the internal lateral ligament. There was a definite defect in the quadriceps extensor tendon above the patella. A roentgenogram

Operation was performed under ether anesthesia. The tendon and patella were exposed by a linear incision. The quadriceps was completely ruptured at the site of its attachment to the patella, no fibers remaining intact. Drill holes were made in the upper edge of the patella and the tendon was brought down and sutured back in place with kangaroo tendon. The leg was immobilized in a plaster cast in full extension for seven weeks. Recovery was complete in five months with complete flexion and extension of the knee and with stabilization so far as the lateral ligaments were concerned (Figs. 5A and B).

The patient was seen again at our request February 6, 1936, nearly three years after the injury. At that time he had full flexion and extension of the knee with a stable knee and no pain or disability. He did have, however, a small defect in the center of the quadriceps above the patella, about the width of a finger which seemed as if the central part of the tendon had pulled loose from the patella again (Fig. 5B).

CASE VI. C. H. F., colored male, aged sixty-two years, was admitted to the Employees' Hospital November 7, 1934, soon after an injury to the region of the left knee. When walking down some steps with a load on his

back he suddenly slipped and while trying to regain his balance he suddenly heard and felt something snap in his knee. He fell to the floor the joint. The patient could not fully extend the leg. There was a definite defect in the quadriceps tendon above the patella. Roent-

A B

FIG. 5. Case V. A. and B. Showing end results after operation for rupture of the quadriceps extensor tendon; practically a full range of motion.

A B

FIG. 6. Case VI. A. and B. Showing the end result after a repair of the ruptured left quadriceps tendon. There is a full range of motion at the knee with no separation at the site of rupture.

C

FIG. 5C. Roentgenogram of Case V showing rupture of the quadriceps tendon and detachment of a small fragment of the patella.

but arose with much difficulty.

Examination on admission revealed a senile individual with definite evidence of arteriosclerosis and hypertension. The left knee was swollen and there was evidence of free fluid in

genogram showed a rupture of the quadriceps tendon.

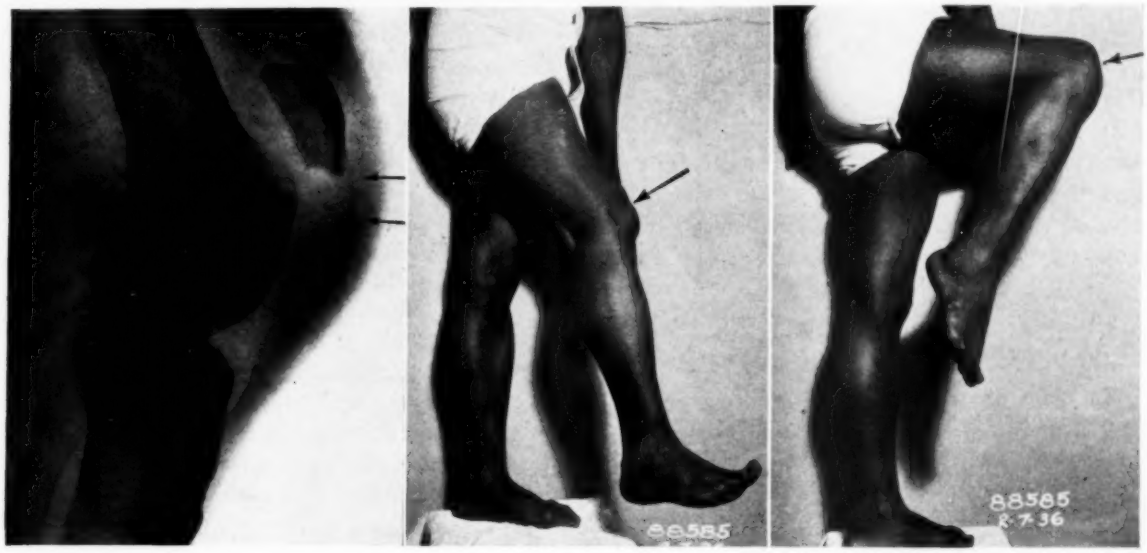
Operation revealed a complete rupture of the quadriceps tendon about 1½ inches above the patella. There was also a tear extending outward on each side, into the lateral expansions of the quadriceps. A few fibers were left intact but were frayed out and stretched. The ends of the tendon were smoothed with a scalpel and united with kangaroo tendon using mattress sutures. The tears in the lateral expansions were sutured with chromic catgut. The leg was immobilized for five weeks in a plaster cast when active motion and massage were begun. Recovery was complete in five months with full motion at the knee. The patient was seen again at our request January 2, 1936. The tendon was intact and flexion and extension were complete. There was no pain or disability (Figs. 6A and B).

COMMENT. Systemic disease could be demonstrated as a predisposing cause in 2 of these cases. Cases IV and VI showed senility, arteriosclerosis and hypertension. Case V did not reveal any definite systemic disease.

RUPTURE OF THE PATELLAR TENDON

Rupture of the patellar tendon is much less frequent than that of the quadriceps. It is produced by the same mechanism, but it is very likely that direct violence is much

more frequently the cause of rupture here, than in the quadriceps because it is more subject to trauma in falls on sharp objects.



A B C

Fig. 7. Case vii, A. Retraction upward of the patella in a rupture of the patellar tendon as revealed by the roentgenogram; B and C. end results after operation for rupture of the patellar tendon. There is practically full flexion with a few degrees short of full extension.

The patella tendon may rupture at any site but it is more frequently at its attachment to the patella or tibial tubercle.

The signs and symptoms of rupture of the patellar ligament are practically the same as that for rupture of the quadriceps. Here, however, the defect is palpable below the patella and the patella is retracted upward. A lateral roentgenogram will usually show the abnormally high displacement of the patella (Fig. 7A). Roentgenograms should be made in order to rule out a gross fracture of the patella, as it was very difficult to do otherwise in the case herewith reported.

Treatment here is early operation just as in rupture of the quadriceps. The quadriceps muscles undergo retraction and fibrosis in an injury of this kind just as they do in fractures of the patella and ruptures of the quadriceps tendons after long neglect. The after-treatment is the same as that given for rupture of the quadriceps.

CASE REPORT

Case vii. J. M., colored male, aged forty-two years was admitted to the Employees' Hospital February 1, 1935, ten days after injury. He slipped while walking on ice and snow and fell on a pavement. He did not know whether the tendon ruptured before he fell or when the knee struck the pavement. He suffered severe pain in the knee and could not walk immediately after the injury. He consulted a physician the next day and was treated for a "sprain" of the knee until he came to the Clinic. The complaint was pain in the knee joint with inability to extend the knee, "having to drag it along, when he walked."

Examination revealed swelling of the knee with inability to extend the knee. There was a hiatus below the patella the width of two fingers. The patella was displaced upward considerably and was more freely movable than normal. Roentgenogram showed the patella to be located abnormally high and also a rupture of the patellar tendon (Fig. 7A). There were no evidences of systemic disease.

Operation revealed a complete rupture of the patellar tendon about one inch below the lower patellar border. The edges were smoothed and the ends of the tendon were sutured together with kangaroo tendon. The leg was immobilized in extension in a plaster cast for four weeks after which time active motion was begun and heat and massage were used. He was advised to report back to the clinic at intervals but he was not seen again until over a year later, February 7, 1936, when he reported at our

request for final follow-up examination. He returned to work, however, in two months after operation. Examination February 7, 1936, over a year after operation showed practically full flexion of the knee with a little less than full extension. There was no complaint or disability, however. The patellar tendon appeared to be slightly elongated and the patella displaced, slightly upward even though there was no defect at the suture line in the tendon, (Figs. 7B and C).

It is likely that this patient returned to work too soon and that too early strain on the newly formed scar caused elongation of the patella tendon.

RUPTURE OF THE CALF MUSCLES

The muscles of the calf may be partially or completely ruptured but partial ruptures are more common. The ruptures here as elsewhere may be caused by direct or indirect violence. They frequently occur in young athletic individuals. Rupture of the rudimentary plantaris is frequent and is commonly referred to as "tennis leg." Ruptures of the calf muscles usually occur during activity such as running or jumping and when the muscles of the calf are in forcible contraction. A direct blow, however, may be the cause. One of our patients was sitting in a porch swing which fell across the calf resulting in a rupture of the gastrocnemius.

Pain usually begins as a sudden sharp stab at the height of action of the muscle. There is continued pain and soreness which is increased on active motion of the foot. In complete ruptures there may be a definite hiatus palpable through the skin. The treatment in incomplete ruptures consists simply of immobilization of the leg in flexion at the knee and plantar flexion of the foot for two or three weeks, with gradual resumption of exercise. Complete rupture, on the other hand, may demand open operation and suture of the torn muscles. There is no doubt that many cases of muscle ruptures in athletes go untreated and are commonly called a "Charlie horse."

CASE REPORTS

The following are cases of ruptured calf muscles which we have operated. We have seen many cases of incomplete ruptures, however, which we have treated conservatively with perfect functional results.

Case VIII. M. T., white female, aged twenty-six years, was admitted to the Employees' Hospital, June 2, 1933, two weeks after she felt a sudden pain in the left calf while walking. She was treated by rest, immobilization and heat for two weeks, with little improvement in symptoms.

Examination upon admission revealed a young woman in excellent condition except for a residual paralysis in the muscles of the left calf with equinus deformity, which resulted from an attack of poliomyelitis when she was about one year of age; however, there was little atrophy of the calf muscles. There was local pain and tenderness to a marked degree over the middle of the calf which was increased by dorsiflexion of the foot.

Operation revealed a rupture of the inner head of the gastrocnemius at the musculotendinous junction and also a partial rupture of the soleus beneath it. The muscles were sutured together with chromic catgut and the leg was immobilized in flexion at the knee and plantar flexion of the foot, in a plaster cast for one month. Recovery was complete with normal motion in the leg after two months. The patient was seen again in January, 1936; there had been no further complications and function was perfect.

Case IX. E. B., a white female, aged thirty-nine years, was admitted to the Employees' Hospital June 6, 1936, complaining of severe pain and disability in the calf of her left leg for six weeks. She was sitting in a porch swing when the swing fell on the flexed contracted leg. She stated that there was a definite depression in the calf immediately following the injury. She had tried home remedies without relief.

Examination on admission showed limitation of motion in the left foot and an old ecchymosis in the skin over the left calf. Any attempt at dorsiflexion, or to raise her weight on the toes was very painful.

Operation revealed a partial rupture of the outer head of the gastrocnemius at the musculotendinous junction, about half of the muscle fibers on this side having been pulled away from the tendon. These were sutured back in place with chromic catgut. The leg was immobilized as in case I and complete recovery was obtained in two months. The patient was again seen at our request in January, 1936,

over two years after the injury; there were no further symptoms and function was perfect.

Case x. A. H., colored male, aged seventeen years was admitted to the Employees' Hospital, April 28, 1936, after having received an injury to the calf of his right leg. He stated that a few hours before admission, when he stepped off of a street car the door was closed too quickly forcing him to jump off. He experienced a sudden severe pain in the right calf immediately upon landing on his toes.

Examination revealed a youth who was well nourished but slender and muscular. There was no evidence of any systemic disease. The right leg was the same size of the left at the calf and there was no visible ecchymosis. There was, however, severe pain and tenderness on pressure over the lower part of the calf muscles which was also exaggerated by dorsiflexion of the foot. Operation revealed a partial rupture of the outer part of the gastrocnemius at the musculotendinous junction. The muscle was replaced to its original site and sutured to the tendon with chromic catgut. Postoperative treatment was the same as in Cases viii and ix. Recovery was complete in six weeks. The patient was seen again in March, 1936, almost a year after his operation; there had been no further symptoms and the end results were excellent.

RUPTURE OF THE ACHILLES TENDON

Ruptures of the Achilles tendon is about equally as infrequent as rupture of the patellar tendon. It occurs almost entirely in middle aged individuals who are very active. The mechanism of production is the same as that of a rupture of the gastrocnemius. The symptoms are pain and loss of ability to extend the foot in the equinus position or to raise the weight of the body as in standing on the toes. In complete ruptures there is a definite hiatus palpable in the tendon at the point of rupture. The foot can be dorsiflexed to an unusual degree. There may or may not be swelling and ecchymosis over the injured area. The belly of the gastrocnemius is retracted unusually high in most cases. Partial ruptures are treated by immobilization in a plaster cast for two or three weeks, but if they are not properly treated they may end in a complete rupture, as in Case xi.

Complete ruptures of the Achilles tendon should be treated by operation and suture of the tendon. Immobilization in a plaster cast with the leg partially flexed at the knee and the foot in the equinus position is necessary for four weeks when active motion can gradually be instituted. Weight bearing in six weeks from the time of injury with a raised heel is advisable. The ultimate end result in these cases is very satisfactory.

CASE REPORT

Case xi. J. H. E., white male, aged forty-five years, tripped on a small rock and fell, followed by immediate pain in the region of the left heel cord. He did not consider the injury very serious and continued his duties even though he had slight pain above the heel when walking. A few days later a small mass appeared above the left heel and slight tenderness persisted. He was not incapicitated, however, and did not consult a physician. About one month following the original injury while running vigorously he developed a sudden sharp pain in the region of the left heel cord and fell to the ground. He was completely disabled in the leg and was admitted to the Employees' Hospital the same day, stating on admission that he had broken his left heel cord.

Examination showed the man to be middle aged, well developed, well nourished and with no evidence of any systemic disease. There was moderate swelling, pain and tenderness in the region of the left Achilles tendon. The belly of the calf muscle was retracted slightly upward as compared to the normal side and the left foot could be dorsiflexed to an abnormal degree even though this was painful. A definite gap, the width of two fingers could be palpated about 3 inches above the heel. A roentgenogram revealed what appeared to be a rupture of the Achilles tendon with separation of the ends.

Operation revealed a complete rupture of the Achilles tendon 3 inches above its attachment to the os calcis. The ends were smoothed over and brought together and sutured with kangaroo tendon. The after-treatment consisted of immobilization in a plaster cast with flexion at the knee and planter flexion of the foot for three weeks. At the end of this time the cast was removed and the foot brought into a right angle position and another cast reapplied for

three more weeks. Active motion, massage and physiotherapy were then begun and the patient was allowed to walk with a raised heel in two weeks with the aid of crutches. Healing and return of normal function were complete in five months. The patient was re-examined January 27, 1936, about three years after operation. There was perfect function with no indication of an elongation of the tendon and there had been no further complications.

HERNIA OF MUSCLES

Herniation of a muscle has seldom been described but probably is very common. According to Haldewan and Soto-Hall[8] "a true herniation of a muscle is characterized by a bulging of the relaxed muscle through a defect in its sheath and the disappearance of this bulge when the muscle contracts." A muscle hernia may be very troublesome causing weakness of the part and pain on motion. Strangulated muscle hernias we believe to be rare and cause more discomfort and disability than any other type. We have seen a case recently which we believe to be of this type, which is reported herewith.

Case XII. E. S., white male, aged forty-five years, on March 20, 1936, was struck on the outer side of the middle third of the right thigh with a twenty pound sledge hammer. He was referred to the Clinic the day following his injury.

Examination revealed an extensive subcutaneous hemorrhage and ecchymosis extending down below the knee. In the middle third of the right thigh on the outer side there was a semispherical swelling about 1½ inches in diameter. The area was very firm, having almost the consistency of bone and was slightly painful to pressure. There was moderate limitation of motion of the leg at the knee and considerable pain in the area on extreme flexion and extension of the knee.

This patient refused operation but there were all evidences of a rupture of the fascia lata and a partial rupture of the vastus lateralis with herniation through the fascia with strangulation of the torn muscle.

Case XIII. E. W., a colored male, aged twenty-seven years, was recently seen in the Clinic for an injury to his arm. Further examination revealed the presence of an old healed gunshot wound over the outer head of the left gastrocnemius, the bullet having made its entrance and exit about 2 inches apart. There was a plainly palpable defect in the fascia over the muscle about 2 inches in length and the width easily admitted one finger. When the muscle was relaxed it bulged considerably through the fascial defect but when contracted, as in standing on the toes, it disappeared. There was no pain or disability. Operation was not advised because of the absence of symptoms.

RUPTURES OF OTHER MUSCLES AND TENDONS

Rupture of many other muscles have been described frequently in the literature, particularly ruptures of the supraspinatous, which were first described by Codman in great detail. The experience of Wilson[9] and Brickner[4] prove conclusively that ruptures of the supraspinatous tendon are much more common than they were once thought to be. Wilson states that in eighteen months previous to 1930 he diagnosed the condition in 10 patients, 7 of whom submitted to operation, which confirmed the diagnosis. Previous to that time he had diagnosed the condition only twice in all his experience. He concludes that the condition is common, which is in accord with the opinion of others who have written on the subject, and reports an original operation for surgical repair.

Rupture of the extensor pollicis longus following fractures of the radius have been reported frequently. Rupture of the extensor tendons of the fingers are very common and are seen often. The triceps, rectus abdominis, muscles of the neck and the pectoral muscles are infrequently the site of rupture, according to the literature.

SUMMARY AND CONCLUSIONS

Ruptures of muscles and tendons are common injuries. They occur most frequently in middle-aged individuals who are physically active but in whom age has begun to weaken the stress and strain which the tissues can tolerate. Disease plays a definite part in the causation but is not a necessary prerequisite by any means.

These injuries are frequently neglected by the patients themselves as well as by the physician. They are usually treated for sprains, bursitis, arthritis or some similar condition. Careful examinations should be made for localized pain, loss of function, painful function of a muscle or a defect in a muscle or tendon with or without ecchymosis. Early diagnosis and treatment prevent disabilities. Complete ruptures should usually be operated and sutured while partial ruptures may usually be treated by immobilization. The treatment, however, in any case must be individualized.

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ORGANIC CALCIUM IN HEALING OF FRACTURES

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IN previous reports the responses of the calcified tissues, i.e., bones and teeth, to various dietary changes were recorded.[1,2] It was found that the fractured fibulae, in rats healed more readily when the animals were on a diet in which protein was present in abundance and especially when calcium and phosphorus were present in adequate quantities and proper ratio.[11]

In these studies two methods of measurement were used, (1) the "breaking-strength" test devised by Lindsay and Howes[3] and used so effectively by McKeown, Lindsay, Harvey and Lumsden,[4] and, (2) the series of histological studies of the fractured bones as described by Downs and McKeown.[2]

Inasmuch as the results from the two methods, that is the "breaking-strength" and histological, to a considerable degree parallelled each other, it was felt that in their joint application a fairly satisfactory method of determining metabolic effects on bone healing and growth had been found.

In the entire series of studies a number of forms of calcium and phosphorus had been used, but eventually the salt mixture of Osborne and Mendel[5] was adopted as being the most completely available at that time. The composition of this salt mixture, based on the mineral content of milk, is as follows:

	Gms.
$CaCO_3$	134.8
$MgCO_3$	24.2
Na_2CO_3	34.2
K_2CO_3	141.3
H_3PO_4	103.2
HCl	53.4
H_2SO_4	9.2
Citric acid and H_2O	111.1
Fe citrate $1\frac{1}{2}$ H_2O	6.34
KI	0.020
$MnSO_4$	0.079
NaF	0.248
$K_2Al_2(SO_4)_4$	0.0245

At some time after these described studies had been published, an organic calcium phosphate, hexacalcium-inositol hexaphosphate (calciphos—Bilhuber-Knoll) made its appearance. Different types of studies, largely clinical,[6,8] suggested that it was more readily and more completely metabolized than were the inorganic forms.

In order to test these favorable clinical results by more exact laboratory procedures, the experiments were continued, following in general the methods described in the earlier reports but with inovations to permit a comparison of the efficacy of various forms of calcium and phosphorus including this organic calcium salt.

The experiment was divided into what may be considered as four separate phases:

A. Weight gains of animals.
B. Blood chemistry.
C. Breaking-strength tests.
D. Histological studies.

Eight groups of 112 animals each were started on experimental diets, after having been standardized on identical basic diets, at two to four week intervals. Each such group was divided into eight subgroups of 14 animals each. The individuals of each subgroup received identical diets and all animals were handled as nearly identically as possible. The age of all animals of a given group was the same, but the age of the animals in different groups varied from twenty-eight days at the beginning of the experiment to four months. Corresponding subgroups of 14 animals each received identical diets.

It will be seen that the total number of animals used was 896 and the average age was seventy-one days. The average age is of importance because of the fact that in all of the tests except the histological, the

Table I

Group	Distribution of Ages and Diets by Subgroups								
	Age	Diet 1	Diet 2	Diet 3	Diet 4	Diet 5	Diet 6	Diet 7	Diet 8
A	28 days	14	14	14	14	14	14	14	14
B	28 days	14	14	14	14	14	14	14	14
C	45 days	14	14	14	14	14	14	14	14
D	45 days	14	14	14	14	14	14	14	14
E	90 days	14	14	14	14	14	14	14	14
F	90 days	14	14	14	14	14	14	14	14
G	120 days	14	14	14	14	14	14	14	14
H	120 days	14	14	14	14	14	14	14	14
Average age.....	71 days	112	112	112	112	112	112	112	112
Total number animals	896								

data was handled by corresponding subgroups, disregarding the differences in age. This was done intentionally so that age variations might not materially affect the final average result for a given diet. In the histological studies comparisons were made only between animals of the same ages and run for identical periods.

Table II

COMPOSITION OF DIETS

Diet 1.	Oz.	Diet 5.	Oz.
Casein	9	Casein	9
Starch	26	Starch	26
Crisco	11	Crisco	11
Cod liver oil	3	Cod liver oil	3
"Calciphos"	2	"Phosphoric acid	1
	—	Calc gluconate"	1
	51		—
			51
Diet 2.		**Diet 6.**	
Casein	9	Casein	4
Starch	26	Starch	40
Crisco	11	Crisco	4
Cod liver oil	3		—
"Calc Phosphate"	2		48
	—		
	51		
Diet 3.		**Diet 7.**	
Casein	9	Casein	4
Starch	26	Starch	40
Crisco	11	Crisco	4
Cod liver oil	3	"Calciphos"	2
Organic Calcium Phosphate from milk	2		—
	—		50
	51		
Diet 4.		**Diet 8.**	
Casein	9	Casein	9
Starch	26	Starch	26
Crisco	11	Crisco	11
Cod liver oil	3	Cod liver oil	3
Calc glycerophosphate	2	Osborne and Mendel salt mixture	2
	—		—
	51		51

The basic diet was the same in all subgroups, being composed of casein, starch and crisco. The salt content varied in the different diets and in the markedly deficient diets 6 and 7, cod liver oil was eliminated, while in all other diets it was present in identical quantities.

All the animals of all the groups, were weighed weekly, at the same time measuring the rates of incisor tooth growth after the method of Addision and Appleton.[9,10]

At varying periods blood was withdrawn from the saphenous vein and tested for clotting time and changes in the cellular composition. In several instances this was followed by withdrawing enough blood for calcium and phosphorus determinations.

All animals were standardized first on their respective diets for a five day period, and then under ether anesthesia following the method of McKeown and his co-workers the right fibula was cut as near its midpoint as possible and the soft tissue wound was sutured.

Beginning twenty-one days after operation, one animal from each subgroup was killed every three days to and including the sixtieth day, and the right hind leg and the mandible were removed.

The legs from alternating animals of alternating subgroups were tagged with identifying numbers and placed in formalin for fixation for histological studies.

The remaining legs were dissected carefully and the bones placed in a desiccator for twenty-four hours until evenly dried. They were then cut and tested for breaking strength of the healing fracture as detailed by Lindsay and Howes.[3] Of the mandibles which were removed, those from the twenty-first day, thirtieth day, thirty-ninth day, forty-eighth day and sixtieth day were fixed in formalin for histological study, and the balance were discarded.

RESULTS

A. Weight Gains. In the case of all of the experimental diets, the very young animals did not take the diets well, and weight gain was abnormally slow and irregular. In the groups of older animals, however, (six weeks and over) with the exception of the deficient diets 6 and 7, the weight increments were at least normal for all diets, and in the case of diets 1 and 3, quite distinctly above those in the control or other experimental diets. Also it is obvious that these weight gains are consistent and regular throughout the course of the studies.

B. Blood Chemistry. Quite accidentally it was noted during the progress of the experiments that in the animals on diet 1 (the hexacalcium-inositol hexaphosphate diet) the blood was darker than in the other animals and clotted immediately upon egress from the vessels.

This phenomenon was checked and it was found that the blood of the animals on diet 1 clotted consistently in less than one minute and formed a peculiarly tough clot, which did not separate from the serum as usually occurs. In all the other diets, including diet 3, the clotting time varied from two to as high as three minutes. In

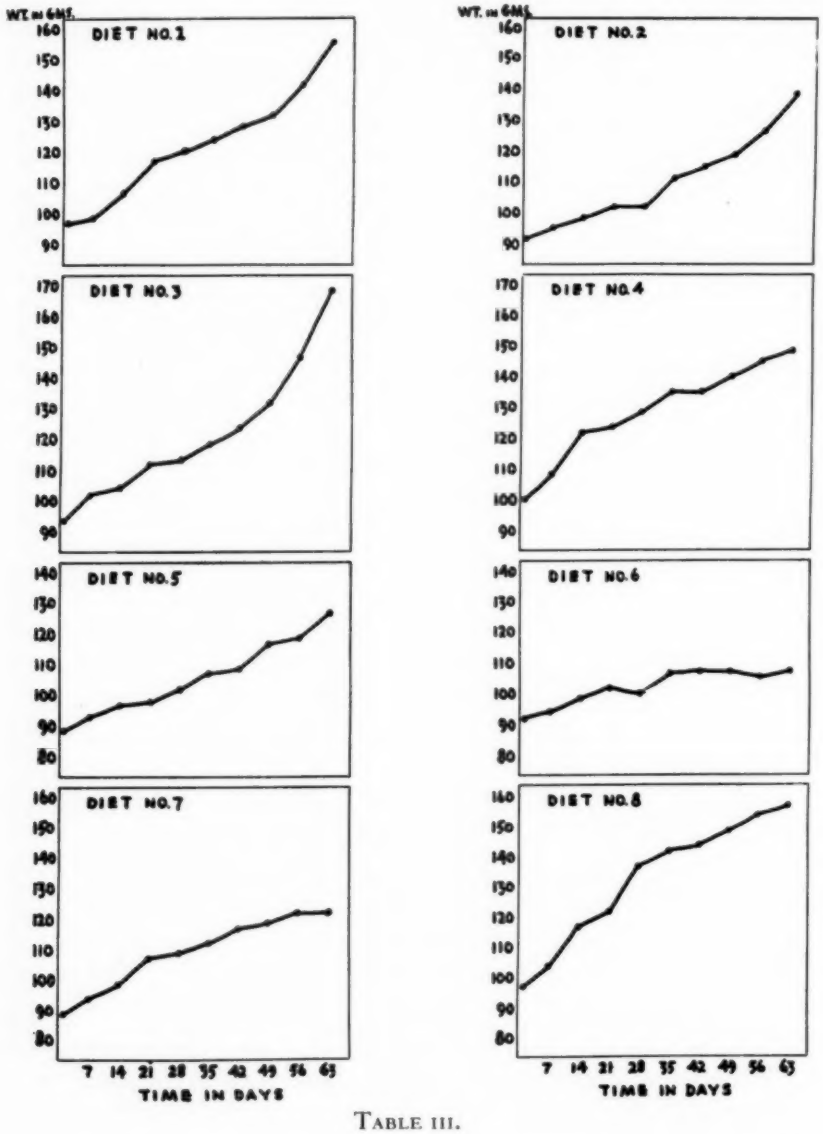

TABLE III.

an effort to explain this variation, cell counts and blood chemistries for calcium and phosphorus were done on a considerable number of animals, but no consistent variation in these conditions could be found. It is quite likely that were it not so difficult to obtain adequate quantities of blood, continued tests would explain the lowered clotting time for animals on diet 1.

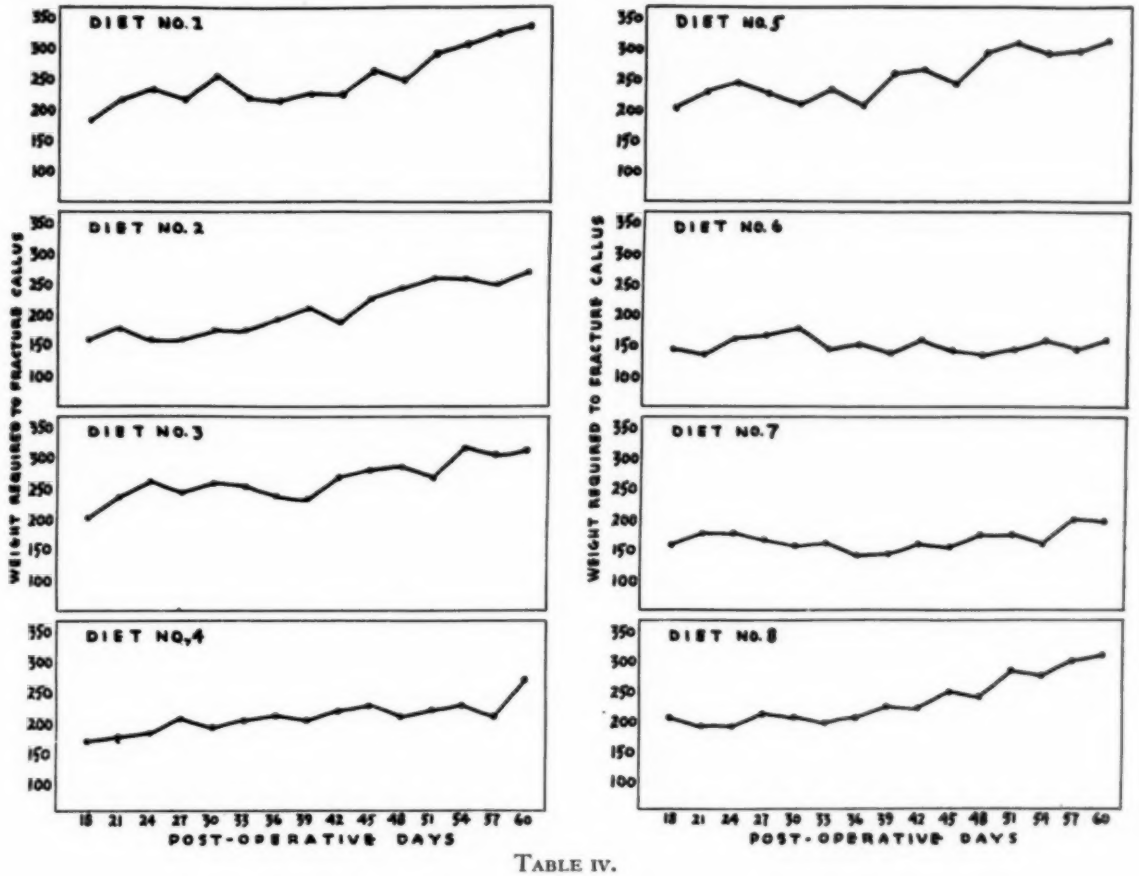

Table IV.

C. Breaking-strength Tests. In general the breaking-strength tests followed similar lines as the results obtained by Lindsay and Howes[3] in that the general direction of the curves was similar, as well as being like those obtained by McKeown et al.[4] Inasmuch as earlier studies had revealed that calcification, or at least true ossification, did not begin until after the eighteenth day, the present series was planned so that these curves do not begin until the twenty-first postoperative day. Judging by the direction and form of the curves, the diets used are of importance in healing of fractured bone, in approximately the following order listed: 1, 3, 4, 5, 2, 8, 7, 6.

The strength of the healed fracture was greater with diet 1 than with diet 3, which contained organic calcium phosphate (diet 1 hexacalcium-inositol hexaphosphate and diet 3 calcium phosphate from milk), were definitely superior in so far as this effect upon bone healing was concerned. Diets 4 (calcium glycerophosphate), 5 (calcium gluconate and phosphoric acid), 2 (dicalcium phosphate) and 8 (Osborne and Mendel salt mixture) while they showed variations, did not differ greatly in the entire results shown. In both diet 6 (no vitamin, no salt mixture) and diet 7 there was very little, if any, healing, as evidenced by the breaking-strength of the callus, although the animals on diet 7 (no vitamin plus calciphos) were healthier and heavier.

It will be seen that these curves do not have the regularity which is shown by those

of Lindsay and Howes.[3] This is because of, (1) the fact that the animals in the present series have a wider age variation than

Fig. 1.

Fig. 2.

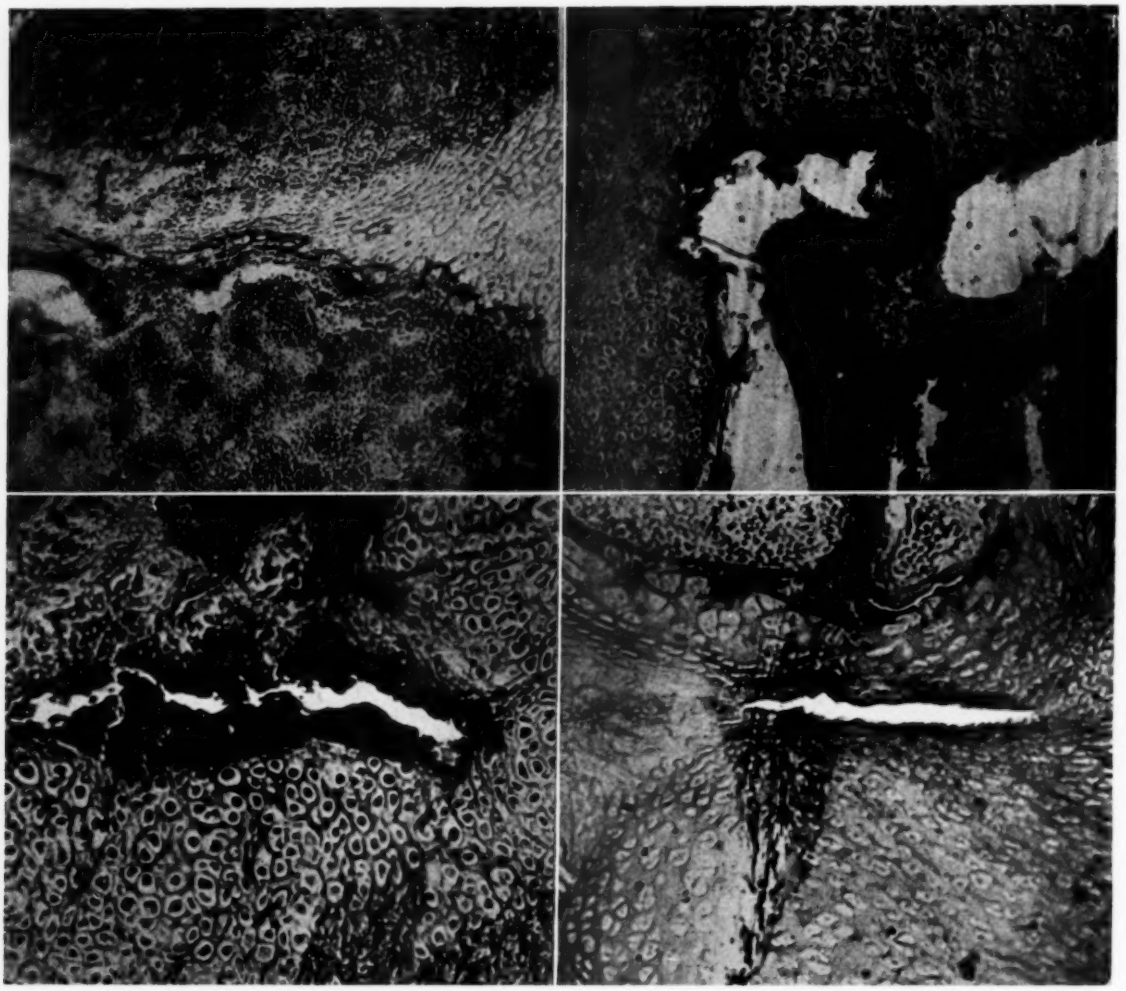

Fig. 3.

Fig. 4.

theirs did; (2) that the basis for the curves in the present series is strict averages, whereas theirs were computed on the basis of correction of the "standard deviation of mean," and (3) the smaller number of animals in the given series.

It will also be apparent that the "breaking-strength weights" in the present series are smaller than was the case for the Lindsay and Howes series. This is undoubtedly due to the younger average age of the animals in the present series.

D. Histological Studies. Since so much depends upon interpretation of histological preparations that it is extremely difficult to rate the various subgroups, one fact was extremely outstanding in the study of these slides. The animals on the two organic calcium salt diets showed a much more "orderly" series of changes than did those on the other calcium and phosphorus rations and of these two series, animals on diet 1 (calciphos) showed the more rapid progress toward the production of a callus of normal bone structure. The changes in the bones of the animals on the organic calcium diets were more advanced at earlier stages than was true of either the other experimental diets or the "controls" on diet 8. Sections of healing fractures of the animals on deficient diets 6 and 7 never showed the picture of normal mature bone as was the case on the other diets, although

those on diet 7 showed some approximation to the normal process of bone repair. This was exactly in line with the results of the breaking-strength tests.

Fig. 5.

Fig. 6.

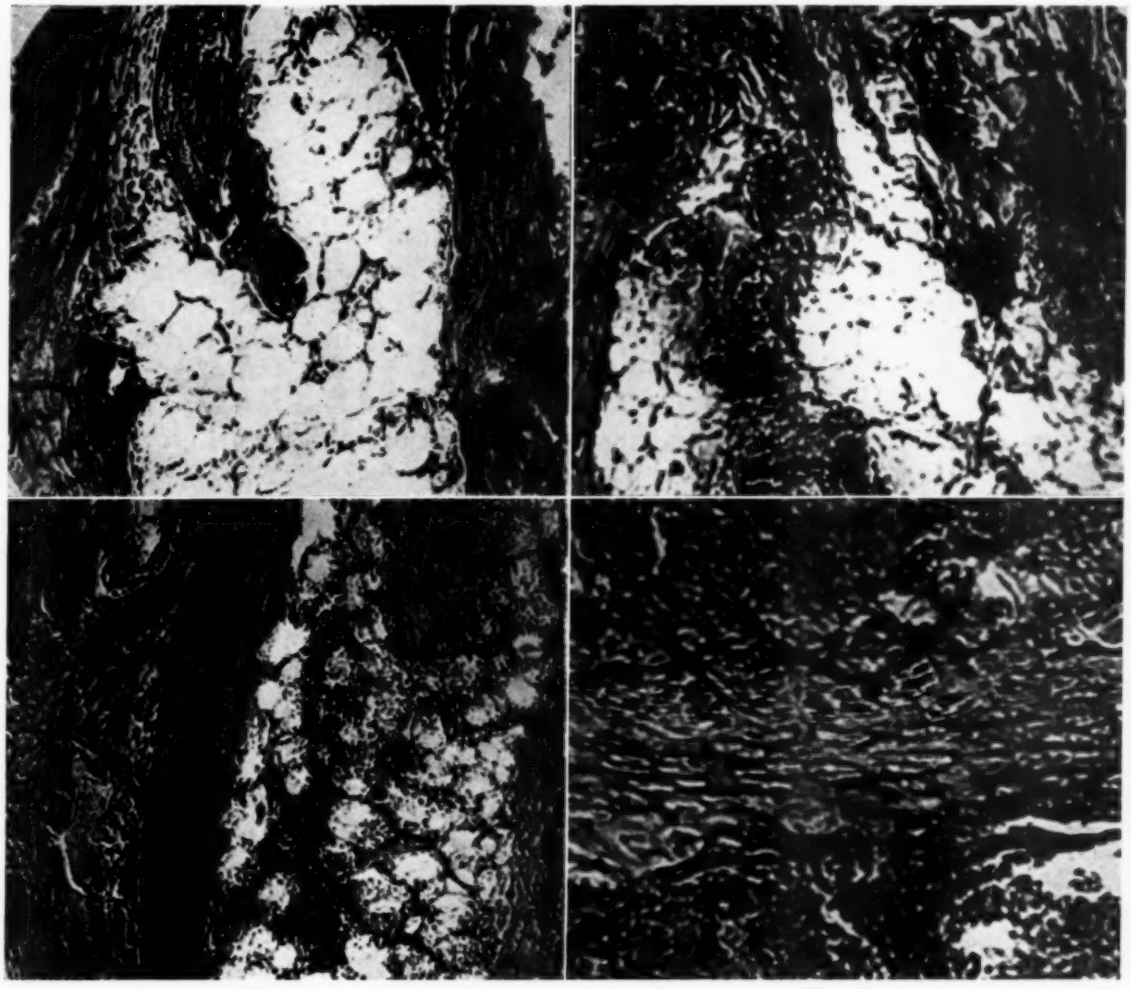

Fig. 7.

Fig. 8.

Animals on diets 1 and 3 showed a quite mature type of bone and by the thirty-sixth day; those on the control diet were at approximately the same stage on the forty-fifth to the forty-eigth day, while the others lagged behind in variable order.

Figures 1 to 4 inclusive are photomicrographs of sections of animals forty-five days old at the beginning of experiment and sacrificed on the twenty-seventh postoperative day. Figure 1 (diet 1) shows very few remaining true cartilage cells with young osteoblasts nearly meeting at the midpoint of the callus line. Calcium is present but is evenly distributed in and among the cells. Figure 2 (diet 3) shows fairly numerous cartilage cells of an older type with depositions of calcium of an earlier type and true bone just beginning to invade the callus. Figure 3 (diet 8) reveals numerous much less mature cartilage cells and extremely heavy deposits of inorganic calcium. Figure 4 (diet 4) shows only large immature cartilage cells, with no or very little calcium deposition and practically no effort at bone formation.

The much later stages shown in Figures 5 to 8 inclusive, are from animals ninety days old at the beginning of experiment and sacrificed on the fifty-fourth postoperative day. They are from the same dietary groups as those shown in Figures 1 to 4

inclusive. Figure 5 (diet 1) shows a return to practically normal mature bone, the only evidence of the fracture line being a slight thickening at that point. Figure 6 (diet 3) lags considerably behind the picture found in diet 1, particularly in a lack of finished "modelling" found in normal bone. In Figure 7 (diet 8) "modelling" likewise lags behind, the marginal bone is not the dense normal type found in either diet 1 or diet 3 and the callus is much thicker. In Figure 8 (diet 4) the cartilaginous callus is considerably condensed, the cartilage cells show a distinct tendency to "line up" but nowhere is there evidence of the formation of mature normal bone and there is still only slight calcium deposition. In each instance these illustrations are quite typical of the results for the groups and subgroups, which they represent. Other diets show an even more retarded state of healing and ossification throughout the sections.

Histological studies of the mandibles of the animals showed nothing of particular moment. No caries was noted in the teeth of the animals on any of the diets, nor did any of the changes sometimes seen in dietary dental studies reveal themselves in dental pulp or surrounding bone.

CONCLUSIONS

The evidence drawn from each phase of the present study indicates that an organic form of calcium phosphate is metabolised more readily than is the case for any of the inorganic forms generally used for such work. While a great variance in the results was not obtained by the feeding of the two types of organic calcium salt, it was also apparent that that obtained from cereal (Indian corn) was utilized more readily by the organism than that from milk.

Satisfactory evidence was obtained to support these conclusions from the weight gains of animals throughout the course of the experiments, from the breaking-strength tests, and from histological studies. Blood chemistry studies were otherwise negative but the more rapid clotting of the blood of the animals fed with calciphos, indicates the value of further work in this direction.

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PLICATION OF SMALL INTESTINE AS PROPHYLAXIS AGAINST ADHESIONS

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LOSS of peritoneal surface of intestines is a frequent complication of a pathological or traumatic process in the abdomen. If the denuded area is left uncovered or improperly treated, adhesions develop which may lead to more serious consequences such as intestinal obstruction, etc. Numerous methods attempting relief have been proposed and reviewed by Ochsner and Garside.[1] The omentum has long been advocated as an excellent substitute for peritoneum, but if the raw surfaces are multiple in number, and particularly in the pelvis, the end result is not always satisfactory owing to the restriction of both gastric and intestinal motility. Recently, amniotic fluid has been tried for the reduction of adhesions by Johnson,[2] Warren,[3] Trusler,[4] and Gepfert.[5] The results of Lacey,[6] however, appear to throw much doubt upon the value of introducing large volumes of such a foreign fluid. Digestive ferments such as papain and pepsin have also been employed to prevent the formation of adhesions by Ochsner and Garside[1] and Yardumian and Cooper,[7] but this subject is apparently still in its experimental stage.

In many abdominal operations, the writer has used a simple procedure to obviate the development of adhesions. The principle is to bring the raw surfaces of adjacent segments of small intestines into apposition and suture them. Since the procedure has been used in a wide variety of cases simplification of description is offered so that the principle is better shown. Three degrees of procedure are therefore described.

1. There is a single rough, denuded or raw spot on the intestinal wall after dissection of the adherent bowel to free it from some pathologic process. The loop containing the damaged area is folded so that the denuded surface is covered by the peritoneum of the adjacent wing of the fold. A running chromic No. 00 catgut is started at the root of the mesentery and carried up to the mesenteric margins of its bowel, approximating the mesentery as shown in Figure 2. The same suture is continued now at right angles along the mesenteric margin of its bowel to the angle of the fold, running around the denuded area as seems best fitted to cover it.

2. There are several damaged areas. After carefully freeing the bowel and mesentery a fold is made as will best cover most raw areas, which usually occur on the same surface. The length of the mesentery will determine the length of the wing of a fold. Ordinarily a wing is from three to four inches long, making each plication include six or eight inches of bowel. In some cases the mesentery is long enough to allow wings of eight inches, or a loop plicating sixteen inches in all. The plication suture is always begun at the root of the mesentery and then continued at right angles along the bowel as seems best. Since the lumen is not constricted by this method, a raw area on the surface of the bowel opposite the mesenteric attachment may be covered with any part of the surface of the opposite wing of the plication. Furthermore, by inspection, no inhibition of intestinal movements ever occurred from such intentional folding.

3. In the presence of very extensive inflammation with marked peritoneal damage, a more radical type of plication is used. If a single plication can not cover all the

raw areas, other plications may be added so that in place of two wings closely approximated by suture there may be three, four, or as many more as needed, each suture to each wing beginning at the root of the mesentery. When necessary the entire length of the small intestines may be plicated. It is in this respect that the folded mesentery and intestines resemble a closed fan.

FIG. 1. Case of complete obstruction lasting four days due to strangulated hernia. Dilated and collapsed bowel shown.

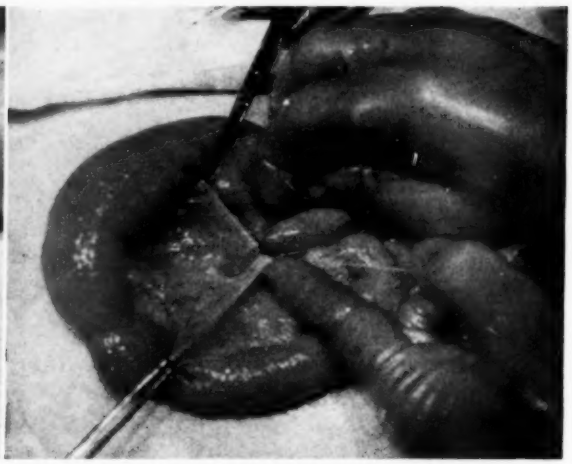

FIG. 2. Loop of bowel proximal and distal to area of incarceration; collapsed gut filling with gas. Plication done to prevent completion of tendency toward intussusception. Photograph shows beginning of mesenteric suture and lines up mesentery to bowel which are to be sutured together.

The illustrations show the plication of one loop of bowel and are chosen for clarity and ease of demonstration of the operative steps.

During the past ten years several hundred cases were treated with intestinal plication. The results have been so consistent that it seems justifiable to make a brief report at present.

The following cases operated during the past two years comprises our latest group of study and can serve to represent the different pathological processes which may be met in practice.

Appendicitis	19
Salpingitis	10
Previous surgery in which gauze packs were used	8
Diverticulitis	4
Hernia	4
Tuberculosis peritonitis	3
Fibroid uterus	1
Apoplectic ovary	1
Ovarian cyst	4
Carcinoma of uterus	1
Infected kidney	1
Intussusception	1

In all cases, including the small series of cases presented and those omitted from other years, there were 3 which had the entire small bowel plicated from duodenum to cecum, and had remained symptom free over a period of six years, two years, and two months, respectively. In 2 cases of strangulated hernia, in addition to plication there was such annular constriction of the bowel that a lateral anastomosis was performed between adjacent wings after plication. This appears much simpler and safer than resection and anastamosis. In the majority of cases simple or single plication of one loop of bowel has been found to be sufficient. Since any condition which destroys peritoneum may eventually produce obstructive symptoms, it becomes obvious that many conditions may require this operation to advantage, such as those exemplified in this series.

The postoperative results have been gratifying. No untoward symptoms have been reported so that there was no individual that needed later operation for relief of adhesions. Apparently, too, there

was no interference with the peristaltic movements because none of these patients have had gastrointestinal discomfort.

Fig. 3. Mesenteric approximation completed. Mesenteric margin of the bowel being sutured; the end of the suture to be at a point under the left thumb in the illustration. The area of incarceration just to the right of the thumb, therefore, is not at the point of angulation.

Fig. 4. Completion of plication viewed from above. Mesenteric suture not visible, being vertical up mesentery to bowel margin. Filling of collapsed bowel well under way through normal peristalsis. Convalescence was uneventful.

Fig. 5. Completed plication viewed from side showing mesenteric stiffening through suture shown in Figure 2. Area of threatened intussusception just distal to area of incarceration shown.

CASE REPORTS

A few brief case histories may further elucidate the uses of plications in our hands.

Case 1. On March 7, 1935, Mr. B. B. was operated for appendicitis through a McBurney incision. The terminal ileum was fixed and flattened along the posterior wall of the pelvis. When it was freed the ventral surface had no peritoneum. Plication covered all the denuded

peritoneum freeing the terminal ileum and mobilizing the cecum.

Case II. On October 26, 1935, Mr. W. C. was operated for appendicitis. Old adhesions involving the ileum fixed it permanently in the pelvis. When these were freed the under surface was so rough that plication offered the means of prevention of reformation of the same situation.

Case III. On March 4, 1934, Mr. R. C. was operated for appendicitis. A McBurney incision was made and the ileum was found to be fixed. Plication offered the only method of covering about six inches of completely roughened peritoneum.

Case IV. On June 10, 1935, Mrs. M. G. was operated for an ovarian cyst and dense adhesions, making the incision over an old midline scar. For denudation of the mesenteric margin of the sigmoid plication was done, a resection being too hazardous. Her postoperative history was normal.

Case V. On April 3, 1932, C. F., a six year old female child, was operated for adhesions from general peritonitis of two weeks standing. A midline incision was made and the entire ileum was plicated. This case was operated during the acute stage because of multiple areas of obstruction. Free pus was present. During the last four years since the operation, there has been no complaint.

Case VI. On March 31, 1934, Mrs. J. M. was operated for loss of peritoneum from intra-abdominal gauze packs at a previous operation through a right rectus incision. The entire pelvic ileum was plicated, one lateral anastamosis being necessary.

Case VII. On February 1, 1936, Mrs. F. N. was operated through a midline incision for adhesions following a salpingectomy and appendectomy. Multiple plications, two and three way, were made throughout the entire small bowel from duodenum to cecum, not six inches of bowel being covered with normal peritoneum before plication. She has gained thirty pounds in weight and is symptom free.

Case VIII. On March 20, 1934, Mrs. M. Q. was operated through a midline scar previously made for a hysterectomy in 1927. She had multiple volvuli with one area of annular constriction with complete obstruction. Multiple plications with lateral anastamoses were made.

CONCLUSION

1. A method had been described for the prevention of adhesions by the plication of the small intestines.

2. In several hundred cases treated by this method, none have developed any symptoms of adhesions.

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BEHAVIOR OF STOMACH AFTER OPERATION FOR DUODENAL ULCER

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DURING the last twenty-five years a great number and variety of operations have been performed on individuals of all ages and both sexes for peptic ulcer. In spite of this, the amount of available information on the motor and secretory functions of the stomach after operations is relatively small and much of it is not altogether reliable. There are several reasons why this should be so. The majority of ulcer-bearing patients who have been operated on are relieved of their symptoms and so pass from medical observation. This is well demonstrated by the fact that most of the studies on patients who have been operated on have been carried out shortly after operation; studies made at a later date are chiefly of patients with recurrent ulcer or some complicating disease. Also, a considerable part of the literature is in the nature of special pleading, and since in such cases there is an obvious bias for or against some particular procedure, the resulting observations are of somewhat less value than would otherwise have been the case.

The present discussion is not intended as a criticism of surgical treatment nor as a plea for any particular type of management; rather is it to be considered in the light of an objective report on some of the phenomena that follow operations on the stomach and duodenum.

It is obviously of some practical importance to know what may be expected in the way of gastric secretion and what changes in the motor properties of the stomach occur after various types of operations, not only because of the influence of these factors on possible recurrences, but also because of their bearing on the applicability of these operations to the treatment of duodenal ulcer. The element of time in the study of changes in gastric function after operation is also important, since countless experiments and clinical observations indicate the essential automatism of the stomach and its tendency to return, so far as the surgical procedure allows, to its original state in respect to both secretory and motor function.

Practically all of the observations mentioned in this paper have been made on patients who were hospitalized on the Gastrointestinal Service at The Mayo Clinic, and the studies represent the work of many men, including Eusterman and Balfour,[3] Walters,[13] Kirklin,[5] Comfort,[2] and Welch.[17] Mention must also be made of the excellent monograph by Meyer-Burgdorff and Schmidt,[7] and of the countless personal and unrecorded observations which have been made by gastroenterologists and surgeons in other institutions and which have been passed on by word of mouth. It should be emphasized that the statements made herein cannot be regarded as final and authoritative, since much additional study will be needed before the picture of gastric function after operation is complete.

Surgical procedures for benign lesions of the stomach and duodenum are of three general types: (1) plastic procedures on the pyloric outlet devised chiefly to widen the pyloric canal, (2) gastroenterostomy, and (3) partial gastric resection with removal of the pyloric antrum and portions of the fundus of the stomach. With the last named procedure, various methods of restoring the continuity of the gastrointestinal canal are in use, the two commonest being the Billroth 1 operation or some modification of it which rejoins the gastric stump directly to the duodenum, and the Polya method of

anastomosing the jejunum laterally to the remaining portion of stomach. Each of these three general types of procedure will be considered separately in respect to the normal response of the stomach in the "cured" case, its behavior in cases in which there are persistent symptoms referable to functional disturbances, and finally, the phenomena produced by recurrent peptic ulcer under the circumstances imposed by the surgical procedure under consideration.

PLASTIC OPERATIONS ON THE PYLORUS AND DUODENUM

Pyloroplasty. The operation of pyloroplasty, with or without excision of a duodenal ulcer, has been widely used, chiefly as a result of the work of Finney, C. H. Mayo, and Judd. It is particularly applicable to the treatment of early duodenal ulcer in young persons, and the results obtained have been usually satisfactory; at least 90 per cent of patients treated by this means reported permanent symptomatic relief. The surgical mortality is low, being about 1 per cent, and convalescence from the operation is usually prompt. The operation reduces the emptying time of the stomach somewhat, but has little effect on the volume and concentration of hydrochloric acid secreted, although the factors of neutralization and dilution by means of free mixture of the gastric and transpyloric juices are probably somewhat increased. This, together with the abolition of the activity of the pyloric sphincter, are presumed to be sufficient to control the mechanisms producing symptoms of ulcer.

There are a small number of patients who, following pyloroplasty, complain of persistent indigestion which may differ greatly from that produced by the original ulcer. The most characteristic things about this dyspepsia are the irregularity of its appearance with respect to the taking of food, the inconsistency of relief from soda and alkalies, and the marked effect of environmental and neurogenic factors. These symptoms often seem to indicate a nervous type of irritability of the gastroduodenal mechanism although in a minority of the cases symptoms may be referable to superficial inflammatory changes in the gastric and duodenal mucosa (gastroduodenitis). In individuals with symptoms of the type just described, acidity will usually be at least as high as prior to operation; roentgenologic examination shows relatively little change except for the deformity produced by the operation and some irritability of the antral segment of the stomach.

Recurrent ulcers following pyloroplasty are relatively infrequent, and, if they do occur, are rarely as serious or troublesome as those which follow other types of gastric operation. They may, however, be extraordinarily difficult to diagnose. The symptoms are usually a duplication of those which existed prior to operation and are often complicated by evidences of chronic penetration, as shown by the projection of pain to the back and a shift in its location to a point near the fossa of the gall bladder.[11] Nausea and vague discomfort immediately after the taking of food are commonly noted and some individuals complain of more or less constant burning distress in the epigastrium or right upper quadrant of the abdomen. Persistent pain at night is common. Large volumes of highly concentrated gastric juice may be obtained on aspiration after an Ewald meal. Since the ability of the stomach to dilute and to neutralize gastric juice is not as greatly affected by pyloroplasty as by other procedures, if a recurrent ulcer forms one therefore expects an unchanged secretory behavior of the stomach. The roentgenologic signs of recurrent ulcer will be considered later.

The appearance of the stomach on roentgenologic examination after pyloroplasty varies according to the period that has elapsed since operation. Early after operation, evacuation of the stomach is somewhat retarded, according to Kirklin,[5] in spite of the enlargement of the gastric outlet. The site of pyloroplasty is easily visualized and, in fact, permanent irregularity of the outlet is produced, the extent of

which depends on the type and extent of the previous operation. Eventually the passage of material from the stomach becomes quite free and the emptying time of the stomach is materially shortened. The intermittent character of gastric evacuation is usually retained, but deformity of the pyloric segment and duodenal bulb become materially less with the passage of time.

It is apparent from this that a recurrent ulcer can hardly be demonstrated in such cases, on the basis of deformity or irritability of the duodenal bulb and the diagnosis must depend almost entirely on the demonstration of a crater. Such defects are often difficult to demonstrate and in some cases repeated examinations may be required. In general the diagnosis of recurrent ulcer after pyloroplasty requires the study and cooperation of both internist and roentgenologist, and careful observation of the patient, on an ulcer regimen, may supplement the information gained by roentgenologic examination.

Lateral Gastroduodenostomy. This operation, first suggested by Jaboulay and later endorsed by Wilkie, is occasionally used as a substitute for pyloroplasty, particularly after disconnection of a gastroenteric anastomosis for stomatal ulcer. As the name implies, the operation is one in which the duodenum and stomach are anastomosed just proximal to the pylorus. The operation has certain obvious advantages and disadvantages. It is easy to secure wide anastomosis, and the gastric juice is then diverted into the duodenum, which is the most acid-resistant portion of the bowel. The deformity of the pyloric outlet produced by the operation, however, adds an element of mechanical difficulty. There may be moderate gastric retention during the immediate postoperative period, and for some time after operation delay in the emptying of the stomach can be demonstrated. However, this mild degree of retention is usually easily controlled by conservative management and the stomach eventually resumes its normal tone. For the foregoing reasons, temporary digestive disturbances may be noted after operation, but these gradually subside as normal physiologic function returns.

The roentgenologic appearance of such a stomach immediately after operation has been well described by Kirklin and others. In one case, in which the patient was examined two weeks after operation, the stomach was atonic and very little barium was passing the pylorus; in the same case, two months later, the edema and inflammation around the anastomosis had subsided and barium could be seen passing through both the pylorus and the new stoma. In this case there was some reduction in free gastric acidity from 114 to 74 units; however, symptomatic results were not particularly satisfactory, neurogenic factors, in this case, being a major problem.

Recurrence or reactivation of an ulcer after gastroduodenostomy is infrequent, but when it does occur it is almost as difficult to demonstrate as is recurrent ulcer after pyloroplasty. The symptoms are likely to be masked at once by recurrence of motor disturbances and delayed gastric emptying, with anorexia, nausea and regurgitation of gastric contents. Pain is atypical and may be either relieved or aggravated by the taking of food. If a penetrating lesion develops, there is frequently a shift of pain to the right upper quadrant with definite posterior projection. Nocturnal distress is common and troublesome. Here again the roentgenologic diagnosis of ulcer depends on the demonstration of a crater, a matter which may be difficult because of the deformity produced by the procedure itself. Persistent motor difficulties may also lead to a suspicion of ulcer even if a crater cannot be demonstrated.

GASTROENTEROSTOMY

The behavior of the stomach following gastroenterostomy deserves consideration in greater detail, since this operation is still the most widely used procedure in the surgical treatment of ulcer. Posterior gastroenterostomy is much more frequently performed than the anterior type. The lat-

ter requires a longer proximal loop and, to provide for the drainage of this segment, enteroanastomosis between the afferent and efferent loops is usually done. The normal results of the operation are well known; the stomach empties rapidly, lower levels of gastric acidity are attained and an active ulcer in the duodenum will heal in a short period. The mere fact that the operation has been so widely employed over so long a period of time is, of course, the best argument as to its merits. It appears to be the most satisfactory procedure for the treatment of cicatricial or obstructing ulcers, or for penetrating lesions with periduodenal and perigastric masses. It may not be suitable, however, for young individuals with pronounced nervous tendencies and large volumes of highly acid secretion.

An opportunity has presented itself for the study of many cases at varying times after gastroenterostomy and the immediate and remote effects on gastric secretion and on the roentgenologic appearance of the stomach are fairly well understood. With a well placed stoma of moderate size, marked and permanent reduction of 30 to 50 per cent in gastric acidity is the rule, as recent studies by Walters[14] demonstrate. It should be emphasized that this reduction in acidity following posterior gastroenterostomy appears to be almost entirely a matter of dilution rather than one of reduction in the amount of acid secreted. The temporary anacidity encountered immediately after gastroenterostomy probably depends on reflex inhibition of secretion. Following anterior gastroenterostomy, the neutralizing and diluting fluids are drained through an enteroanastomosis, and the reduction in gastric acidity is therefore much less striking.

The roentgenologic appearance of the stomach immediately after gastroenterostomy is of considerable interest and it illustrates the fact that great importance should be attached to the postoperative care. As Kirklin[5] has noted, the stomach is often somewhat atonic and contains more or less secretion. The suture line around the stoma may be visible, the adjacent rugae are poorly defined, and the passage of barium through the stoma is not particularly free. If the same stomach is examined several weeks later a very different picture is obtained. The normal gastric tone has returned and the stoma permits the rapid passage of large amounts of barium. The reduction in emptying time after gastroenterostomy is a highly individual matter and no standards can be set for it. If the stomach is completely filled with barium during the course of the examination, rhythmic emptying through the anastomosis can be demonstrated. This appears to depend on the intermittent contraction and relaxation of the jejunum rather than on the development of an anatomic sphincter at the stoma. Usually, small amounts of barium pass the pylorus also, but the bulk of the gastric content escapes through the anastomosis. The stomach usually appears smaller after operation and the gastric rugae in the vicinity of the stoma appear to be accentuated. Peristalsis appears less marked, but it is possible to demonstrate the presence of peristaltic waves through to the pylorus as in the case of the non-operated stomach. Some fixation of the stomach is often noted, but both the afferent and efferent portions of the jejunal loop are usually movable and show the usual markings of the valvulae conniventes. Kirklin[5] emphasized the fact that even after many years the stoma does not tend to close except as a result of marginal ulcer or other disease.

Malfunctioning Gastroenteric Anastomosis. There are some patients who, following gastroenterostomy, will continue to have temporary, mild digestive distress, especially after dietary indiscretions. These symptoms are usually most marked immediately after operation; they are caused by delayed gastric evacuation and decrease with the passage of time. The patient who has a truly malfunctioning gastroenteric anastomosis presents an entirely different picture. In general, faulty gastric motility following gastroenterostomy can be attributed to: (1) an error in the original

diagnosis, most commonly leading to the performance of gastroenterostomy for functional vomiting, (2) a high lying stoma, or one of insufficient size, (3) too long a proximal loop, (4) the placing of the stoma too near the pylorus, (5) a short proximal loop with consequent kinking and traction which interferes with the normal emptying of the stomach, and (6) adhesions which may obstruct the proximal or distal jejunal loop. The symptoms produced by any of the foregoing factors may be considered under one head, and they include anorexia, bloating and belching, epigastric distress after eating, nausea, regurgitation, and, in extreme cases, the vomiting of large quantities of retained gastric contents with subsequent relief. Gross mechanical obstruction cannot be detected roentgenologically in much more than half of the cases. In such cases the gastric acidity is usually low or at least within normal limits, which is in conspicuous contrast to the high levels usually associated with gastrojejunal ulcer.

Roentgenologic examination of the stomach is of great value in demonstrating the nature of motor difficulty of this type and it is frequently possible for the expert examiner to determine exactly why the anastomosis does not function as it should. Too great stress should not be laid on the direction of the proximal loop which may be peristaltic or counterperistaltic, and yet give perfectly satisfactory results.

Another type of motor dysfunction following gastrojejunostomy, which is fortunately rarely seen, has been described under the title of "dumping stomach." The manifestations of this syndrome are chiefly intestinal and are described with difficulty by the patient. Nausea, sweating and prostration are noted after the taking of food and there is often associated diarrhea. The nutritional state of the patient may be impaired because of the fear of taking food. Such complaints are most often noted: (1) after unnecessary gastroenterostomy in the presence of constitutional asthenia and an atonic stomach, and (2) as a manifestation of intestinal hypermotility of nervous origin which may have been apparent before operation. The so-called vicious circle in which the ingested food is regurgitated from the proximal loop into the stomach is rarely seen nowadays and, in fact, a malfunctioning gastroenteric anastomosis from whatever cause is rapidly becoming a rarity.

The management of patients with malfunctioning gastroenteric anastomoses is a most difficult problem and a decision as to the best mode of treatment should be deferred until the symptoms which existed prior to operation have been thoroughly reviewed. The criteria for disconnection of a gastroenteric anastomosis seem to be three: (1) persistent gastric retention, (2) impaired nutrition, and (3) greater discomfort after operation than existed before.

Recurrent or Reactivated Ulcer after Gastroenterostomy. The great criticism which has been directed against gastroenterostomy has been based on the frequency with which gastrojejunal ulcer develops. These lesions appear to result from the lack of resistance to acid aggression of the jejunal mucosa. How frequently gastrojejunal ulcers form has never been satisfactorily determined. The figures given depend somewhat on the criteria used for diagnosis and on the length of time elapsing after operation. Several large series of cases have been studied postoperatively; they seem to establish a rate of occurrence for gastrojejunal ulcer of approximately 3 per cent. Other writers who favor more radical procedures for the treatment of duodenal ulcer report a much higher incidence. A more satisfactory conclusion could be reached if there were some agreement as to what constitutes a recurrence or if one knew how much time should elapse after operation before all danger of recurrence is past. Recurrent stomatal lesions develop in some cases years after the original operation. Other patients have minor inflammatory or erosive lesions in the vicinity which produce symptoms and signs for a brief period and then disappear; others may

have solitary or widely separated hemorrhages as the only symptom. In still another group, the roentgenologic appearance of the stoma may suggest recurrence but there may be no symptoms or other signs to justify such a diagnosis of recurrence.

The type of these recurrent ulcers and the time required for their formation deserves some comment. It is generally agreed that most recurrent ulcers form and give symptoms soon after the primary operation; many form at a very early date, as has been demonstrated at secondary operations for other causes, and practically all have become active within a year and a half. Many of the recurrent lesions have a marked tendency to hemorrhage and penetration, and the formation of inflammatory perianastomotic masses is common; both complications may be present even if the original ulcer has shown no such tendencies. The commonest site for gastrojejunal ulcer is in the first 4 cm. of the efferent jejunal loop and the next most frequent site is the stoma; the afferent loop is only infrequently involved. Most of the ulcers are small, 1 cm. or less in diameter, and solitary lesions are the rule. The penetrating character of the lesion and its location often favor penetration into the transverse colon. There is often associated gastrojejunitis or jejunitis and, in fact, this condition may occur alone without frank ulceration.

The symptoms which are produced by anastomotic ulcers are often conspicuously irregular:

1. Periodicity and relief from taking food and alkali frequently are lacking.

2. Control under medical treatment is difficult.

3. There is a shift in the site of pain. The patient may originally have had pain in the epigastrium or just to the right of it; the pain of the recurring ulcer is lower in the abdomen, often in its left midportion. Projection downward is common. The question of the location of pain in ulcer of various types and its projection has been studied in detail by Rivers[11] and those interested are referred to his article for details.

4. The tendency to hemorrhage of patients who had not previously bled is very striking and often leads to a correct diagnosis.

5. Unusual mechanical and motor phenomena, such as evidence of physiologic block at the stoma and heightened gastrointestinal irritability, are occasionally seen. These phenomena may be attributable to local foci of inflammation in the wall of the bowel.

6. There may be local signs of peritoneal irritation and, occasionally, the formation of palpable masses at the site of anastomosis.

The gastric secretion in the presence of gastrojejunal ulcer shows the widest possible variation depending on the rate at which the stomach empties and on its previous secretory capacity. With active gastrojejunal ulcer, high acidity and evidence of slow or insufficient dilution and neutralization of the gastric juice is commonly found, in fact, the maximal volumes and concentrations of hydrochloric acid are found in such cases. While some individuals may have a gastrojejunal ulcer with low or moderate values for acidity, the mere fact that the gastric acids are persistently high should lead one to keep the patient under observation because of the danger of recurrent ulcer developing.

The roentgenologic appearance of an ulcer at or near the site of a posterior gastroenteric anastomosis is quite characteristic and should lead to a diagnosis in from 75 to 90 per cent of cases. Kirklin[5] found that in about two-thirds of these cases a niche or crater can be demonstrated. In most of his cases he noted that the lesion was in the vicinity of the stoma at the internal gastrojejunal angle, the niche being demonstrable as a marginal projection or as a central fleck. Almost as significant and quite commonly associated with a niche is a localized deformity in the region of the ulcer. With an ulcer at the stoma the gastric rugae may reveal distortion, or there may be marked

swelling of the mucosa and some obstruction to the outlet. A smooth funnel shaped appearance of the stoma is also highly suggestive of ulcer. When the niche is situated well away from the stoma in the jejunum, the markings of the valvulae conniventes may be effaced in this region.

The secondary roentgenologic signs of gastrojejunal ulcer are almost equally important. These include partial closure of the stoma or marked stenosis of the jejunum, with retarded evacuation of the stomach and hyperperistalsis. The jejunum may show similar changes above the site of obstruction. Tenderness over the region of the deformity is common and the mobility of the parts may be lessened. Positive errors in the diagnosis of gastrojejunal ulcer are relatively uncommon but the roentgenologic appearance may be simulated by local spasm, vigorous peristaltic contractions, or local adhesions. For this reason Kirklin[5] felt that secondary signs of obstruction are not a sufficient basis for a diagnosis unless some actual deformity or crater can be demonstrated.

Recurrences at the site of an anterior gastroenteric anastomosis have one rather remarkable feature which has been commented on by surgeons and roentgenologists. Under these circumstances the ulcer may be symptomless until of a very large size. There is often much local edema at the stoma and there may be large accessory pockets which form as a result of perforation.

Gastrojejunocolic fistulas are said to form in about 10 per cent or less of cases of gastrojejunal ulcers of long standing. The symptoms of the condition are variable in the extreme. A number of patients have been seen at the clinic whose symptoms were minimal and no more than could have been accounted for on the basis of an uncomplicated ulcer. In others, bleeding, fecal vomiting and diarrhea immediately after the taking of food have directed attention to the condition. Diagnosis depends on roentgenologic demonstration of a fistulous connection between the stomach and colon which is not necessarily perceived on roentgenologic examination of the stomach alone; frequently a barium enema is required for the demonstration of such a fistula.

In a few remarkable cases, first described by Fairley and Kilner,[4] profound nutritional disturbances with diarrhea, hypocalcemia and tetany were produced by such a fistula. I have recently had the opportunity of seeing one patient who presented an almost typical picture of sprue in association with gastrojejunocolic fistula. The explanation is obvious; the small intestine is completely short circuited by such a fistula and no opportunity is afforded for the absorption of such important substances as vitamins and minerals. I have known of one case in which the diagnosis was made on the basis of the patient's edema, emaciation and pallor. Only a single question was asked, "Have you had a gastroenterostomy performed?" An affirmative answer was all that was required for a diagnosis and for the institution of successful treatment.

PARTIAL GASTRIC RESECTION (BILLROTH, POLYA, ROUX)

The practice of performing partial gastric resection for duodenal ulcer has been the most hotly discussed surgical procedure of our times. The principal argument used in favor of the procedure is that it reduces gastric acidity more than any other operation and that recurrences are almost entirely eliminated. First done extensively on the Continent it has found favor with certain American surgeons and undoubtedly has a place in the treatment of gastric and duodenal lesions, although its application may not be so general as was first proposed. The procedure is certainly of value in the treatment of the ulcer forming patients who have had conservative surgical treatment without obtaining relief, in the treatment of individuals with both gastric or duodenal ulcers or with marked gastritis and in the treatment of certain hyperactive young males with primary ulcers and with

very large volumes of secretion and high free acidity. In general, it has been the procedure of last resort in many cases of ulcer after medical management and conservative surgical treatment have failed. While the mortality is somewhat higher than is the case in conservative surgery, the end results have been excellent in properly selected cases.

The immediate clinical sequela of partial gastrectomy are not as widely known as they should be. The operation itself is an extensive one, no matter what technique is employed, and the period of postoperative disability is correspondingly prolonged. The capacity of the stomach is considerably reduced by the operation, thus complicating the problem of postoperative dietary care. Anorexia, nausea and fullness after meals are common postoperative complaints but they usually disappear in a relatively short time. In a few cases the rapid passage of food into the jejunum produces a sense of intestinal uneasiness with nausea, sweating and prostration after meals. Once the jejunum becomes accustomed to receiving fairly large volumes of food from the stomach this complaint disappears. Certain German authorities claim that the jejunum dilates distally to the point of anastomosis and forms a sort of after-stomach, "Nachmagen," but this has not been a conspicuous feature in cases which have been observed at the clinic. In some cases after radical resection there have been a host of indefinite digestive symptoms which the Germans have grouped under the convenient term of "Beschwerden," and which have been attributed, on the basis of gastroscopic studies, to gastritis. In a relatively few cases macrocytic anemia develops owing to the loss of the intrinsic hematopoietic principle furnished by the stomach. It should be emphasized that most of the complaints which follow partial gastrectomy are of a distinctly temporary nature. The patients should be warned about them and carefully instructed with regard to diet. The patient should be warned not to go too long without eating and should not eat too much at one time until the gastrointestinal tract has had an opportunity to accustom itself to the new state of affairs.

Gastric Secretion after Partial Gastrectomy. The study of gastric secretion after partial gastrectomy is one of extraordinary complexity due chiefly to the large number of variables which enter into the problem. Among these one must particularly consider: (1) the amount of stomach resected and the secretory capacity of the remaining portion, (2) the altered mechanics of the stomach and duodenum which may allow for free mixing of gastric and duodenal juices and also for regulation of the rate and degree of such mixing, (3) the inhibition of gastric secretion incident to jejunal or duodenal overfilling, (4) the factor of time after operation, and finally, (5) the state of the gastric and duodenal mucosa as an absorbing and secreting surface.

The wide variations in acidity after gastric resection recorded in the literature give evidence of the many and diverse methods of study employed as well as of the obviously preconceived ideas of many investigators who have studied the subject. For instance, two early advocates of partial gastrectomy reported that in about 75 per cent of clinical cases there is complete and permanent anacidity after this operation; the most optimistic recent estimate gives a considerably lower figure. Anacidity occurring in the period immediately following operation probably depends in part on reflex inhibition of gastric secretion. There is a great deal of clinical evidence to show that permanent and complete control of gastric acidity is not accomplished in more than 50 per cent of cases by gastric resection. Certainly not enough of the fundic mucosa is removed to cut off the source of hydrochloric acid, since as Watson showed experimentally, even extensive partial fundusectomy does not abolish the secretion of acid. Failure of the operation to produce permanent anacidity is usually attributed to too conservative and limited resection; this explanation is part and parcel of the

widely advanced theory which ascribes a hormone producing activity to the mucosa of the pyloric antrum, and holds that this section of the stomach is essential to the second or chemical phase of gastric secretion. The experiments of Priestley and Mann,[9] as well as the earlier studies of Portis and Portis,[8] would appear to refute conclusively such an explanation. The notion that a nodal center governing gastric secretion is present in the lesser curvature and that its removal affects the production of acid, likewise have fallen under a weight of physiologic evidence. It seems that in the human subject the effect of wide gastric resection on secretion is neither so great as the earlier investigators claimed, nor so limited and temporary as recent experimental studies on dogs appear to show. The clinical observations reported by Walters[13] and Comfort,[2] using histamine as a stimulus to obtain pure gastric juice, indicate that although partial gastrectomy of many ulcer bearing human stomachs greatly reduces gastric acidity, there is little evidence that the actual capacity of the fundic portions to secrete acid is abolished or even materially reduced.

The factor of simple dilution, especially as it pertains to admixture of duodenal juice with gastric content, has certainly been underestimated, and it undoubtedly plays a very large part in the anacidity and subacidity which follow late after partial gastrectomy. The observations of Klein, Aschner and Crohn[6] indicate that a higher percentage of instances of anacidity is encountered in the late postoperative period after the emptying time of the stomach becomes rapid, than in an earlier stage; roentgenologic observations indicate that the motor mechanism of the stomach and duodenum, while temporarily disturbed by resection, tends to stabilize and assume a normal rhythmic character within variable periods of time after operation. It appears that the last word has not been said concerning either the immediate or the late effects of partial gastrectomy on the secretory functions of the stomach of man; continued observation and study of clinical cases in which this operation has been performed are essential to the ultimate solution of the problem.

Roentgenologic Appearance of the Stomach. A series of careful observations on the partially resected stomach have been made by Kirklin.[5] He notes that immediately after operation the line of suture is somewhat irritable and the passage of barium somewhat retarded. The remnant of the stomach may be somewhat dilated and may contain some secretion. At a later date, after complete restoration of motor tone has taken place, the mixture of barium leaves the stomach freely but in a rhythmic manner, apparently being restrained by contraction of the duodenum or jejunum, depending on what type of operation has been performed. Notwithstanding this, the evacuation time of the stomach is greatly reduced and is considerably shorter than that which prevails following gastroenterostomy. Irregularities at the suture line may persist but are of little significance. After a Polya resection the gastric remnant will become funnel shaped with rapid but not continuous evacuation. There is some roentgenologic evidence to show that efficient function is regained more rapidly after a Polya operation than after a Billroth 1 operation.

Symptoms of Recurrence after Gastrectomy. The incidence of recurrent ulcer after gastric resection has been put at a very low figure. Berg[1] in 1927 stated that he had not known of a single recurrence in 400 cases. However, Weir[16] was able to collect 57 cases of recurrent ulcer and he has analyzed the symptoms in some detail. In the majority of his cases the symptoms of recurrence developed within a year after operation. He noted that in a considerable number of cases the onset of symptoms was acute and severe with local pain, tenderness and rigidity. The usual relationship to the taking of food were preserved and the pain was most often described as of "gnawing" or "burning" type. Pyrosis, nausea and vomiting were common accompaniments of

the pain and it was often very distressing at night, requiring frequent feedings and, in extreme cases, opiates. The usual periodicity of ulcer distress was lacking in most of his cases and the story was that of continuous distress. The left upper portion of the abdomen was the favorite site of the pain which was more diffuse than the pain of the primary ulcer. Projection to the back or lower part of the abdomen was frequent and in a few cases there was projection to the chest and left shoulder. In one of his cases there was local perforation with development of abscess; in another, perforation occurred into the colon, and in several more, there was a local penetrating process with involvement of the diaphragm. Gross hemorrhage occurred in about 25 per cent of Weir's cases but obstruction was uncommon for obvious reasons. In only 5 cases in this series of 57 was complete achlorhydria present, and in 3 of these the symptoms of ulcer were very mild and were controlled readily by medical management. In general, a rough parallelism between the level of gastric acidity and the severity of the symptoms was demonstrated. In most of Weir's cases it was a fairly simple matter to make a diagnosis of recurrence from the history and physical findings, in fact, it appears that very little confusion is possible between the symptoms of functional disturbances that follow partial gastrectomy and those that are due to recurrent ulcer.

The roentgenologic diagnosis of recurrent ulcer after partial gastrectomy is a somewhat difficult matter and requires considerable experience on the part of the roentgenologist. Diagnosis depends chiefly on the demonstration of a crater or niche; the secondary signs of obstruction are most frequently lacking. It is important for the roentgenologist to know something of the type of the operation performed in order to make a proper appraisal of the condition in these cases. Knowing the type of procedure it is possible for the expert roentgenologist to demonstrate a crater in a high percentage of cases.

SUMMARY AND CONCLUSIONS

It has repeatedly been emphasized that gastrointestinal symptoms which follow operation for peptic ulcer always deserve a careful hearing. It is apparent that many of the symptoms which occur during the immediate postoperative period are attributable to poor tonus of the stomach, delayed evacuation and minor mechanical difficulties which are of a transient nature. There is also likely to be temporary inhibition of the secretion of hydrochloric acid which may of itself be sufficient to produce some distress. Most, if not all, of these symptoms can be relieved by proper adjustment of the diet and even if this is not done spontaneous improvement is the rule.

The principal diagnostic difficulties arise in cases in which symptoms persist or in which discomfort develops late after operation. Such symptoms may be purely functional, or may be attributable to preexisting gastrointestinal disease. Careful review of the preoperative and postoperative history, study of the gastric secretion on stimulation with histamine and during periods of distress, examination of the stools for blood and repeated roentgenologic examinations are often necessary before a correct conclusion can be reached.

The value of studying the patient in the hospital while on a strict ulcer regimen can hardly be overestimated; there is no other therapeutic procedure which so clearly distinguishes the character of the symptoms arising in cases in which the stomach has been operated on for peptic ulcer. This affords an opportunity for aspiration of the gastric content when the patient is having pain and it may frequently reveal gastric retention which would otherwise pass undetected.

It must be remembered that the chances of temporary functional disturbance are much greater than those of recurrent ulcer and that definite proof should be required before a diagnosis of recurrence is made. This is particularly true of symptoms that arise shortly after operation. The patient

who presents himself with symptoms which have developed long after operation is somewhat more likely to have a recurrence, no matter how long the interval may be or how bizarre the complaints.

The whole matter may be summarized as follows:

1. There is an adequate physiologic explanation for many of the complaints that follow immediately in the wake of gastric surgery for benign lesions. Most of these complaints are attributable to motor dysfunction, which is either self-limited or is amenable to treatment.

2. A temporary reduction in gastric acidity may follow almost any type of surgical procedure on the stomach; however, the stomach tends to resume its normal secretory capacity eventually and it seems that most of the observed reductions in acidity are largely a matter of dilution and neutralization by transpyloric juices.

3. The symptoms of recurrent inflammatory ulcer after any type of operation may be masked by motor and secretory disturbances and are often extremely bizarre. For this reason, extremely careful study may be required to prove the presence of such a lesion.

In a study of these cases it is important to recall that peptic ulcer is not necessarily a local disease and, as Rivers[10] says, "a recurrence of ulceration remains an active threat as long as the factors responsible for such erosion of tissue are unchecked."

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TOTAL GASTRECTOMY FOR CANCER

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TOTAL gastrectomy offers the only curative measure for cancerous lesions involving a large part of the stomach. Its scope of application is, however, decidedly limited. Relatively few cases of extensive malignancies of the stomach present conditions sufficiently favorable to permit undertaking such a radical procedure; the operation itself is attended with major risks; and the experience of operators in general has shown that the procedure is associated with high mortality. Yet, because in an occasional case offering favorable conditions, total extirpation of the stomach has afforded the patient several years of life with comfort, the operation must be recognized as holding an important place in the treatment of extensive cancer.

OPERATIVE TECHNIQUE

The important steps in the operative technique are as follows:

1. Abdominal exposure
2. Exploration of the abdomen
3. Determination of operability of the growth
4. Division of the duodenum
5. Freeing of the stomach
6. Anastomosis of the esophagus and jejunum
7. Closure
8. Provision for early feeding

Anesthesia. Anesthesia, as in every prolonged and serious operation, is a decidedly important factor. Its choice and administration should preferably be in the hands of a trained physician anesthetist. Spinal anesthesia is to be desired wherever feasible, although inhalation anesthetics, notably ether, may be suitably administered.

Operative Steps. Step 1. Abdominal Exposure. A good access that makes the deep seated cardiac orifice available is essential. Either the midline incision, or an incision splitting the upper left rectus muscle, is satisfactory in most cases. The incision should be long, extending from the xiphoid process to well below the umbilicus. In some cases, and especially in the obese patient, better exposure is obtained by cutting the left lateral ligament of the liver and rotating the left lobe of the liver toward the right.

Certain operators, with a view to facilitating the esophagojejunal anastomosis, have suggested paralyzing the left diaphragm by crushing the left phrenic nerve, done a day or two before the operation through a small incision in the neck. The procedure is probably rarely necessary.

A combined abdominal and thoracic approach has been used by some surgeons. Hilarowicz raises a large angular flap of the anterior-inferior part of the left thorax, sectioning the ribs in the process, but leaving the diaphragm and the anterior pleural cavity untouched. Marwedel also exposes the cardia by raising a flap of the lower, left thorax without opening the pleural cavity. His incision is more sparing than that of Hilarowicz and he sections only the 7th, 8th, and 9th ribs. Lobenhoffer produces an artificial pneumothorax before sectioning the lower ribs and raising the thoracic flap. Ohsawa reports an extensive approach in which the pleura is opened and the diaphragm sectioned to unite the pleural and abdominal cavities in one operative field.

The use of the thoracic approach and the sectioning of the ribs increase the chances of pulmonary complications. Pneumothorax, injury to the thorax, and pleural infection are some of the dangers. Trauma to the ribs by interfering with the breathing increases the possibility

of the development of pneumonia, especially in older persons.

Step 2. Exploration of the Abdomen.

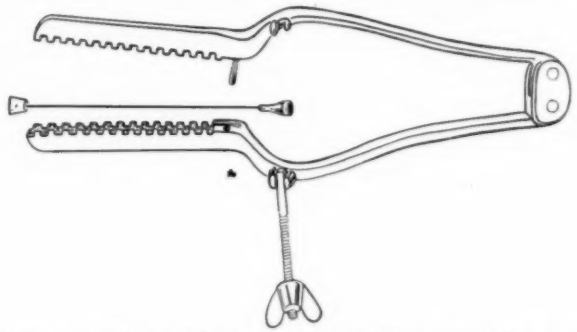

Fig. 1. Author's (H. M. Clute) modification of Furniss clamp. Hinge at proximal end of clamp permits needle to be passed for aseptic closure of duodenum.

The abdomen must be explored for metastases to the liver and to the glands in the gastrohepatic omentum. Total gastrectomy is definitely contraindicated in the presence of metastases to the liver. In cases with metastases to the gland-bearing area the operation may be undertaken, but the advisability of proceeding with it is questionable.

Step 3. Determination of the Operability of the Growth. The growth is next inspected. There must be a margin of good tissue either at the upper end of the stomach or at the lower end of the esophagus in order to ensure a secure anastomosis. The surgeon should actually feel this good tissue before proceeding with the operation. In the past, the writers have made the mistake of starting the operation, and after section of the duodenum, have found that the malignant area extended up the posterior and lateral sides of the esophagus so that no good tissue was available for anastomosis.

Step 4. Division of the Duodenum. If resection is decided upon, the first step is the freeing of the duodenum at the pylorus. The blood supply is clamped and cut, and the fingers are inserted into the lesser peritoneal sac. The pylorus and the duodenum are separated from the pancreas and fully mobilized.

The duodenum is clamped with the writers' modified Furniss clamp (Fig. 1), care being taken that the edge of the duodenum comes just to the distal end of the clamp. A stout clamp is placed

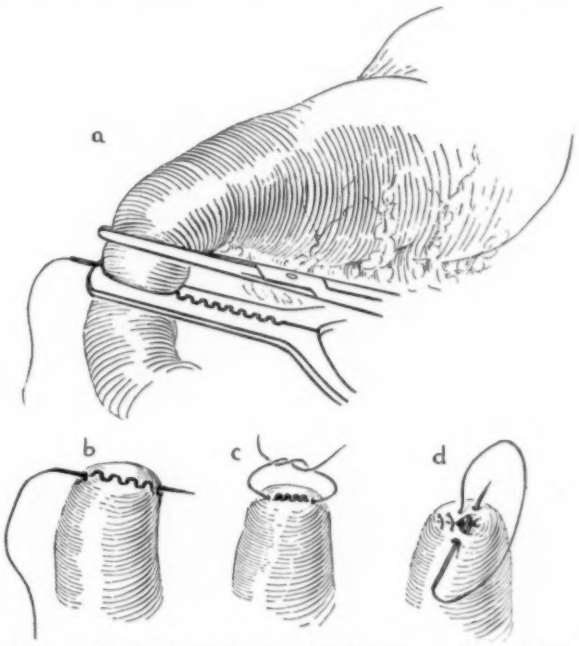

Fig. 2. Method of closing duodenum with modified clamp, using straight intestinal needle instead of pin to hold duodenum after division from stomach. Tying the suture closes the stump without soiling, and inversion can then be completed.

on the distal stomach. A needle carrying catgut is inserted from the distal end of the clamp to hold the duodenum, and it is then divided with the actual cautery, and the clamp removed (Fig. 2). The needle is drawn through the duodenal stump and the suture tied. The stump is turned in and closed with either continuous or interrupted mattress sutures. A covering of omentum is applied with a few interrupted sutures.

Step 5. Freeing of the Stomach. The omentum is freed from the greater curvature of the stomach with a series of hemostats, which are placed over the vessels that are readily seen. These vessels are then tied. The gastrohepatic omentum is divided with multiple hemostats, care being taken to keep as far as possible from the stomach and to include as many glands as possible.

The stomach is mobilized by ligating the right gastric artery, the left gastric

artery, and the vessels on the greater curvature side. All tissues are cleaned away from the esophageal end of the stomach

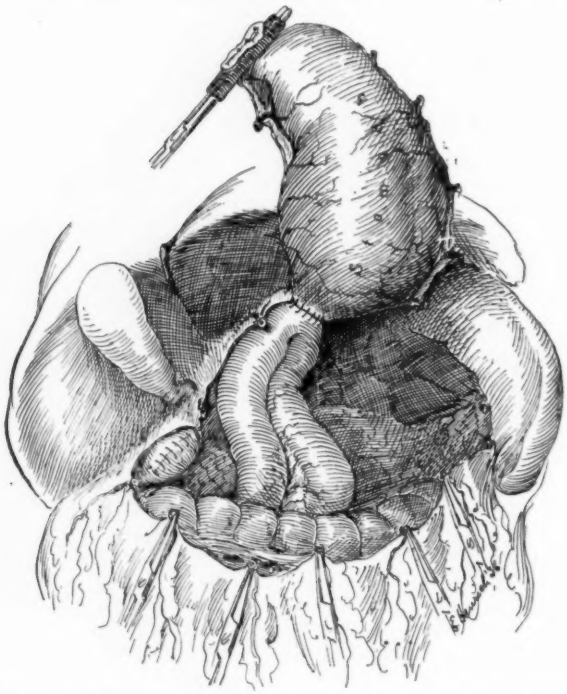

Fig. 3. Duodenum closed but not yet covered with omentum. Gastrohepatic and gastrocolic omenta separated from stomach and vessels tied. Stomach drawn strongly upward to expose esophagus. This traction facilitates placing the sutures between the esophagus and long jejunal loop. The anastomosis may be retrocolic as shown or antecolic.

down to the peritoneum, but care is taken in this process to preserve carefully the small arteries and veins on the esophagus. The stomach, which is now free and hanging by the esophagus, is retained for use as a retractor during the anastomosis.

Step 6. Anastomosis. Esophagojejunal Anastomosis. The transverse colon is lifted up, exposing the mesocolon. A small incision is made through the transverse mesocolon, care being taken to avoid the midcolic artery. The ligament of Treitz is identified, and at this point the curve of the jejunum is picked up, and a long loop, of a foot or more, is carried up through the hole in the transverse mesocolon to the esophagus. The proximal loop of the jejunum which will lie between the ligament of Treitz and the esophagus should be free, that is, it should not be loose or tight.

The stomach is pulled forward out of the abdomen. The jejunal loop is carried up to the posterior wall of the esophagus and fixed by means of stay sutures in the position where the anastomosis will be established. A posterior layer of sutures, of either fine chromic catgut or silk, is placed (Fig. 3). It is important to start suturing on the jejunum fairly close to the posterior side of the mesentery, in order to have plenty of free jejunum left over in front to complete the anastomosis.

Transverse incisions are made across the posterior surface of the esophagus and into the lumen of the jejunum. Soiling is prevented by the use of suction as the viscera are opened.

A second layer of running, over-and-over sutures is placed between the jejunum and the posterior wall of the esophagus. When the side of the stoma has been reached, the incision in the esophagus is carried slightly farther, then more sutures are placed, and this cutting and suturing are continued until the stomach has been entirely cut, leaving only a tiny hole to be invaginated.

A second and outer layer of sutures is placed between the anterior surfaces of the esophagus and the jejunum. A few more interrupted sutures are placed to reinforce the anastomosis, especially at its lateral angles, and to prevent the pull of the jejunum from causing any strain on the esophagus (Fig. 4).

Throughout this entire procedure no clamps are used on the esophagus or jejunum. Only occasionally is a right angle clamp used on the stomach to prevent discharge of the contents. The use of clamps in this operation is, in fact, a hindrance; they are clumsy and they prevent working in a deep hole. Furthermore, when clamps are not used, any vessel that bleeds can be seen and controlled.

Suction is used throughout the entire procedure to prevent soiling from the stomach contents. Sometimes gauze is placed in the side of the stomach to check leakage.

Variations in the technique described will be found in the work of other operators. A few surgeons recommend lifting up the jejunal loop in front of the transverse colon, instead of carrying it through the transverse mesocolon. The writers have never carried out this method. It would seem that the posterior treatment is the better since it is shorter and does not necessitate the additional anastomosis between the proximal and distal loops of the jejunum that is required in the anterior treatment.

Another variation in the technique is found in the particular method of suture. Some operators use more than two layers of suture; others prefer the Murphy button to suture; and still others use clamps. Various forms of reinforcement of the suture are also in use. Hilarowicz sheaths the esophagus with the intestinal tube formed by anastomosing the jejunal loops, thus permitting the true anastomosis to be concealed in the interior. Coenen performs an esophagojejunal anastomosis through a so-called Y-shaped invagination method. He raises the uppermost jejunal loop through a slit in the mesocolon, makes a transverse section of the loop, and reduces its circumference to that of the esophagus. The loop is then slit for a few centimeters and the esophagus is invaginated in the loop. End-to-end anastomosis of the oral and aboral loops of the jejunum is then done.

Esophagoduodenal Anastomosis. While esophagojejunal anastomosis is the method preferred by most surgeons, owing to the fact that the insufficient length of the esophagus and duodenum seldom makes union of these organs possible, there are a few operators who believe that such union is always practical through systematic mobilization of the esophagus and duodenum. Such lengthening of the esophagus involves separating it at the diaphragm, sectioning the vagus nerves, and producing traction on the esophagus by means of the fingers, or the retained stomach, forceps or clamps. Von Haberer reported a successful case of esophagoduodenal anastomosis in a patient with ulcer, and the patient lived six years. Beresow found

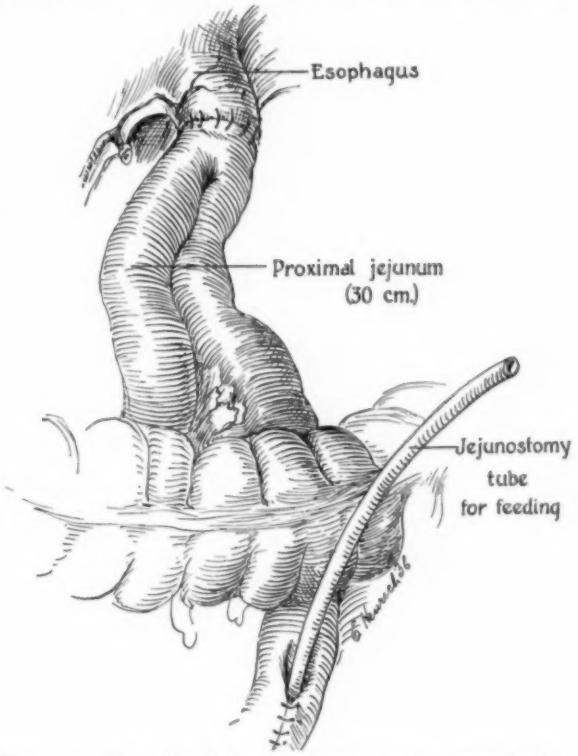

Fig. 4. Esophagojejunal anastomosis completed. Lateral anastomosis of the jejunal loops not necessary in the retrocolic operation. A jejunostomy for feeding is a desirable addition.

the procedure practical in 2 cases. Very few anastomoses of this nature have been performed, so it is difficult to judge the results. Certainly, the traction on the esophagus endangers the tissue. In the rare case, such as that reported by Balfour and Gray, in which the abdominal portion of the esophagus was found to be abnormally long, esophagoduodenal anastomosis may be possible, and in such a case it should be undertaken.

Creation of a Gastric Pouch. Still other operators have suggested providing a gastric pouch for the reception of the food (Marwedel, Hoffmann, Roeder, and Percy). The pouch is formed by uniting the jejunal loop below the anastomosis. The writers do not believe that the formation of such a pouch is necessary.

Step 7. Closure. The hole in the transverse mesocolon through which the je-

junum was carried must be carefully sutured around the distal loop to prevent herniation at this site. These sutures must not penetrate the lumen of the gut and they must not constrict it.

The abdomen is closed in layers. Before the suturing of the separate layers, through-and-through sutures of double silkworm gut, placed about ¾ of an inch apart, are passed through all the layers of the abdominal wall for tying later after complete closure. A series of short running stitches of No. 2 chromic catgut is used to close each layer. If the patient is obese, it is an advantage to insert a rubber dam drain in the superficial fat. If there is any question of the completeness of the closure of the duodenum, a drain may be inserted at that site through a separate stab wound.

Step 8. Provision for Early Feeding. In order to permit healing of the anastomosis, no food or liquid is taken by mouth for four days. One of the best means of providing for early feeding is to insert a catheter in the distal jejunal loop at the time of the operation, bringing the tube out through the omentum. Some operators slip a Levine tube down the esophagus at the time of the operation, and when the anastomosis has been completed it is passed into the distal jejunal loop. To force a tube down the throat into the loop in an unconscious patient is difficult, and the procedure may cause soiling of the operative field. If the tube is to be used, it can usually be more readily passed postoperatively after the patient becomes conscious.

GENERAL CONSIDERATIONS

Case Selection. The first consideration must be the general condition of the patient. A person who is thin and preferably under fifty years of age is the ideal case. One of the writers (H. M. C.) has carried out his most successful total gastrectomy in one such patient, who was a woman of slender build, about thirty-six years of age. The fat, plethoric patient, on the other hand, does not withstand the operation well, and the technical difficulties encountered in carrying out the procedure in this type of patient are great.

As to the influence of the type of growth upon the success of the operation, reports in literature have shown that favorable results have been obtained from total gastrectomy performed for several different types of cancer. The linitis plastica type of growth, however, lends itself best to operation for several reasons. Although the growth is extensive, it develops mostly at the pylorus, leaving the cardiac end free, and it is less likely to be complicated with distant metastases.

The operation is definitely contraindicated in the presence of metastases to the liver. In cases with metastases to the omentum, when the involvement is not too extensive the operation may be performed successfully, but the advisability of its use in these cases is always to be questioned.

Preoperative Care. Most of the patients who seek relief are in poor condition; they are anemic and weak, and the kidney function may be disturbed from lack of fluids. Blood transfusions should be given for the anemia a day or two before the operation. Glucose and fluid administrations will improve the kidney function and provide for some restoration of the liver function.

Postoperative Care. Morphia is used to control pain, but care should be taken to avoid interference with respiration by administering too large doses of this drug.

The fluid intake is maintained by an intravenous administration of 10 per cent glucose solution in 750 to 1000 c.c. of normal salt solution once a day, and by subcutaneous administration of 1500 c.c. of saline solution once a day. Because of the discomfort caused by the subcutaneous administration, this is done in the morning, and the intravenous is given at night.

If there is a jejunal tube in place, the Murphy drip apparatus may be used, and drops of peptonized milk and water are continuously passed into the jejunal tube at the rate of 30 to 40 a minute.

If there is vomiting, distention or distress, a Levine tube is passed and left in place in the jejunum.

After four or five days, sips of lukewarm water are given by mouth for twenty-four hours. Then, a thin gruel of oatmeal or cream of wheat, an ounce or two an hour, and water are given, followed by a soft diet of milk toast, custards and cream soups. After ten days a semisolid diet may be taken without difficulty. For a time the patient continues to eat five small meals a day, but gradually he adapts himself to normal, regular eating.

Complications. Pulmonary complications are indeed serious and should be especially guarded against. To avoid hypostatic congestion the patient should be turned frequently in bed, that is, every half hour, and deep breathing should be encouraged. Coughing will help to empty the lungs. Oxygen as well as carbon dioxide may be used, although their use has not been demonstrated to lessen the complications materially. As a routine measure the patient is now placed in an oxygen tent for the first twenty-four to thirty-six hours after the operation.

There is always the danger of the development of infection. If this appears in the wound it is not serious. Intra-abdominal and subdiaphragmatic infection may result from soiling during the operation, from a leak in the anastomosis, or from necrosis that is the outcome of a stitch pulling too tightly.

Paralytic ileus, postoperative intestinal obstruction, hemorrhage, pulmonary embolus, and wound disruption must be recognized as possible complications in order to deal actively with them, as the indications arise.

Troublesome postoperative achlorhydric diarrhea may be controlled by the administration of fifteen drops of dilute hydrochloric acid three times daily.

Patients have also complained of soreness of the tongue and insides of the cheeks. In the writers' cases the glossitis seemed to be associated with an anemia, and after treatment for the anemia the tongue inflammation disappeared.

Anemia has been found to develop in practically all cases of total gastrectomy. A few observers claim that it has been of the pernicious type, but the majority have noted a mild secondary anemia. A noteworthy characteristic of the anemia, whether of the pernicious type or the secondary type, is the lateness of its development. One would expect that with the deprivation of the products of gastric secretion from the digestive process anemia would develop early. Experimental studies on gastrectomized dogs have also brought out the fact that the anemia develops late (Fontès, Kulin, Thivolle; Ivy, Morgan and Farrell). In dogs the anemia has been of the hypochromic type. From the experimental studies some observers conclude that reserves of hematopoietic materials must exist in the body, and it is only when this supply becomes exhausted that anemia develops. Ivy, Morgan and Farrell are of the opinion that digestion following total gastrectomy, although subnormal, is adequate to provide for absorption of hemoglobin and red cell building materials, and it is only when the state of digestion is reduced, or when an extraordinary demand is made upon it, that anemia develops.

A disturbance of the acid-base equilibrium apparently takes place following the removal of the stomach. In the observations that have been made in this connection, no change in the acidity of the urine after meals has been noted as in the normal person.

Only a few studies of the utilization of foods following the operation have been made. It would seem that the proteid and carbohydrate metabolism is little affected,

but that the absorption of fat is somewhat disturbed after the operation.

End Results. From the standpoint of the duration of life, it cannot be said that the results of total gastrectomy are gratifying. Death has occurred in many cases from recurrence or metastasis. When there has been no recurrence, the development of anemia seems to have been the factor responsible for death. The longest period that a patient has lived, according to reports in medical literature, is four years and eight months. This was a case reported by Zîkoff. Mayo's patient lived four years.

TABLE OF END RESULTS OF TOTAL GASTRECTOMY FOR CANCER
1930 to March, 1936*

	Date	No. of Cases	End Result
Atkinson and Masson	1934	1	Case reported only to 26th day after operation; patient well.
Bull and Stang	1934	1	Well and working 13 months after operation.
Clute and Mason	1930	1	Lived 3½ years.
Judd and Marshall	1930	1	Well 9 months after operation.
Lobenhoffer	1934	1	Lived 6 months.
Morrison	1933	1	Living 4 months after operation.
Ohsawa	1930	1	Well 45 days after operation.
Percy	1934	2	1 patient well 5 months after operation. 1 patient lived 1 year.
Roeder	1933	1	Well and working 3½ months after operation.
Shima	1931	3	1 patient lived 10 months. 2 patients lived 14 months.
Stahnke	1933	3	1 patient lived 7 months. 2 patients well 2 years after operation.
Streicher	1934	1	Well 10 months after operation.
Von Haberer	1930	1	Living 1½ years after operation.
Walters	1933	2	1 patient lived 10 months. 1 patient well at 2 years.
Wangensteen and Carlson	1931	1	Well 10 months after operation.

* Reports of the following writers were not available: Bence, G. *Orvosi betil.*, 79: 1162, 1935; Blumental, N. L. *Sovdt. klin.*, 19: 940, 1933; Bull, P. and Stang, J. *Norsk Mag. f. laegevidensk.*, 96: 165, 1935; Charrier, A. and Chauvenet, A. *Bordeaux chir.*, 3: 262, 1932; Kaplan, A. V. *Vestnik kbir.*, 24: 270, 1931; Lange, K. *Chirurg.*, 2: 1117, 1930; Rosenthal, N. and Abel, H. A. *Tr. Am. Gastro-Enterol. A.*, 36: 215, 1933; Ungley, C. C. *Newcastle Med. Jour.*, 12: 192, 1932.

The accompanying table covering the majority of total gastrectomies for cancer reported between the years 1930 and 1936, although not an accurate analysis because of the lack of final reports, nevertheless presents a fair picture of the periods of survival in cases treated within the past few years.

CONCLUSIONS

From the standpoint of restoring health to the patient for a period of several months or years, the results of this operation are decidedly favorable. The patients have good appetite, they enjoy eating, and the disturbances subsequent to the operation may be controlled sufficiently to avoid discomfort. Some patients are able to resume their occupations and lead active lives. One of the writers' cases (gastrectomy performed by H. M. C.) lived three and a half years with comfort and was able to return to her work.

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POSTOPERATIVE LUNG ABSCESS*

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CONFLICT still exists concerning the relative frequency of postoperative pulmonary suppuration, its etiology, and its proper treatment.

That pulmonary suppuration has become more prevalent in the last three decades is manifested by the attention it has received by the hospital, the clinician, and by the voluminous literature which has appeared in recent years.

The material for this paper is drawn from a review of the literature and from clinical observations of patients at Knickerbocker Hospital, The Fourth Division Surgical Service at Bellevue Hospital, and the Surgical Service at Willard Parker Hospital.

For two reasons no experimental laboratory work has been done. First, the literature is replete with excellent experimental data, some of which has been done by recognized, painstaking and unprejudiced laboratory workers. Second, the average clinician rarely can conduct a laboratory research and draw his conclusions entirely from facts so established. All too frequently he has a preconceived idea which he wishes to substantiate by experimental data, and so unconsciously distorts the results.

Two main theories have been advocated: (1) The aspiration theory, where the abscess is supposed to be caused by aspiration of infected material from the upper air passages into the lung via the bronchus, and (2) The embolic theory, where the abscess is initiated by emboli lodging in the lung parenchyma. The issue becomes more and more controversial as one proceeds with a literary review, and more conflicting since the same data or similar data are used to prove the opposing theory.

In looking through the literature, we find the aspiration essayists numerically in the ascendency. Whether this is to be taken as a proof of the theory or as a mere Coué repetition, is an open question. It certainly is a plausible theory.

How can lung abscess be explained more easily?

1. Bacteriologically, it has been demonstrated that practically all chronic lung abscesses are infected with the same flora that may be found in the patient's mouth, about the teeth and in the tonsils.

2. Bronchoscopically, it has been shown following operations for tonsillectomy, both under local and general anesthesia, that pus, blood and corruption have found their way into the upper bronchi (usually not demonstrable below the bifurcation). This supposedly follows any operation.

3. Cultures of this aspirated material actually demonstrate organisms that are often found in lung abscess.

4. The right lower lobe is more frequently involved due to its straighter bronchus.

5. Suspensions of material removed from the teeth and tonsils have been introduced into the bronchi of experimental animals, resulting in the formation of lung abscess in these animals.

In the presence of these facts, one is almost forced to conclude that the aspiration theory has passed the theory stage and has become a fact. Yet, the proponents of the embolic theory, though having fewer essayists, present data which cannot be ignored.

1. The relative ease and the constancy with which lung abscess can be produced in animals by freeing infected emboli in the venous blood stream;

2. That infarction in the lung, in minor or major forms, is a relative frequent postoperative accident; (found by Cutler and Hunt[1] as 1 in 50);

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3. That when infarction occurs, it is relatively more frequent in the lower lobes than in the upper, and appears more often in the right lower lobe than in the left (a ratio of 4 to 3);

4. That thrombosis is much more frequent in operative areas that are actively or potentially infected;

5. That embolus is more frequent where movement of the parts cannot be avoided, i.e. movement of the throat in talking, chewing and swallowing. Also, operations just below the diaphragm, where the act of respiration causes constant movement.

Experimentally, it is relatively easy and constant to produce lung abscess in dogs by infected emboli. Van Allen et al.[2] used sputum containing fungus Nocardia for intrabronchial insufflation in 5 dogs. None developed lung abscess, while with the same material introduced via embolic innoculation into the lungs of 5 dogs, all developed lung abscess.

Likewise, Cutler and his associates have demonstrated the ease and constancy with which lung abscess may be produced by infected emboli. For a number of years they have done careful experimental work on this problem. Due to the uncertainty and difficulty of producing lung abscess experimentally by the bronchogenic route, they soon abandoned the aspiration theory. They have, however, produced lung abscess at will by introducing emboli in various ingenuous ways into the blood stream.

Their first efforts were far removed from a natural process found in man. Blood was withdrawn from the experimental animal and mixed with bacteria of a given dilution. It was allowed to clot outside the body and a small portion was then placed in a capsule, or a segment of excised vein, together with a small lead shot. This "embolus" introduced into the jugular vein, formed an infarct in the lung, the presence of the shot permitting immediate localization by means of an *x*-ray film. By this means the development of the pulmonary abscess could be studied radiographically during its entire course.

They have, from time to time, modified their technique in each instance, attempting to simulate the natural formation of an embolus.

They isolated the jugular vein of an experimental dog and allowed an abscess to develop, which in time prepared the vein for the site of an infected thrombus, and also developed an active immunity in the host for the particular organism or organisms used. The vein was then traumatized and the blood flow temporarily obstructed, causing the formation of a thrombus. Emboli were eventually dislodged into the blood stream from this thrombus without ever opening the vein or adding anything to the blood stream to cause clotting, or mechanically putting the organism into the blood clot. With this technique they produced lung abscess.

It is generally recognized that if experimental lung abscess is produced by any one of the ordinary pyogenic bacteria, it will be of minor importance and will readily heal.

Considering the production of experimental lung abscess as described by Cutler and the mentioned fact, I cite the case of F. B.

Case 1. This man was recently admitted to the Knickerbocker Hospital for observation because of a sudden severe pain in his right chest followed by expectoration of dark, bloody sputum. On examination, an incised granulating wound was found on the left lateral aspect of his neck. When questioned, he stated that he had had an abscess at this site, which was incised one week prior to admission. It was further noted that there were many veins in the immediate area.

Chest examination revealed a pleural friction rub in the right side, slight dulness over this area, together with pain on deep inspiration. The temperature during the ensuing week was 102°F. The patient subsequently developed pleural effusion and 800 c.c. of fluid were withdrawn. Culture of this fluid showed no growth.

Subsequent *x*-ray examination revealed a small lung abscess with a fluid level. The original cough had subsided and at no time did the abscess cause a productive cough, since it

never ruptured into a bronchus. In fifteen days it had healed beyond *x*-ray identification. This is quite in conformity with experimentally produced lung abscess, where pyogens initiate the process.

Consideration of this mentioned case prompts a pertinent question; viz., if lung abscess is caused by an embolus from an operative field, why does the lung abscess flora simulate the mouth flora rather than the type of organisms found in the original wound from which the embolus originated? The answer is that if cultures could be made in the incipient stage of such an abscess, they would show the same organisms found in the operative wound, but as soon as the abscess ruptures into a bronchus, contamination with mouth organisms takes place and this is responsible for the typical chronic, thick walled abscess that is usually seen in man. Supporting the fact that this contamination can readily take place, I cite an observation made on patient T. B., on whom a lobectomy had been performed.

Case II. Following lobectomy, he developed a large bronchial fistula, which could be visualized through the chest wall wound. It had been noted on two occasions that, following a nose cold, the discharge from the fistula, which had been serous, promptly became purulent. In the midst of his convalescence, he developed a very angry infection of his throat, cultures of which demonstrated a streptococcus. A diagnosis of scarlet fever was eventually made. Although the fistula had been closing rapidly just prior to this infection and the discharge had become scanty and serous, in eighteen hours the fistula stoma became swollen and inflamed and cultures from this source demonstrated a similar growing and staining streptococcus to that found in the throat.

This bronchial contamination has also been demonstrated experimentally. Van Allen produced lung abscess in a dog with an embolus carrying Staphylococcus albus which, by itself, produces essentially an insignificant and rapidly healing abscess. But, when he caused sputum organisms to be insufflated following the formation of such an abscess, the virulence and chronicity of the abscess were found to be similar to that characteristic of abscesses of the lung initiated by sputum organisms.

If this theory, i.e., the initiation of the abscess by infected embolus and propagation by bronchogenic contamination be accepted, then

this would account, in the embolic theory, for the disparity often existing between the bacterial flora of the abscess and that of the operative field; and, again, why abscess of the lung regularly contains organisms native to the mouth in spite of the excessive difficulty with which these organisms, when introduced into the bronchus, produce abscess of the lung. Thus, the main obstacle of each hypothesis taken alone is removed by combining the two. This mode of origin corresponds, also, with the rarity of the abscess of the lung as a postoperative complication since postoperative embolism is unusual; only certain of the emboli carry organisms capable of forming abscess, and of the abscesses a part only would be expected to acquire communication with a bronchus to permit contamination.[1]

Although there has been no animal experimentation for this paper, a special clinical investigation has been made. This study was followed in the wards of Willard Parker Hospital, where many cases of diphtheria are treated. It seemed to me that if aspiration of the mouth organisms is the common etiological factor producing lung abscess, then we should find somewhat conclusive proof of this theory in cases of laryngeal diphtheria, especially in intubated cases or in patients having paralysis of the larynx. Under such conditions the laryngeal reflex is just as completely removed as when it is abolished by deep narcosis. The result of this investigation was entirely negative as no cases of lung abscess developed following intubation or laryngeal paralysis. To indicate the extent to which this mucopurulent membranous mixture containing mouth organisms penetrates, 100 autopsy protocols were reviewed, showing its presence in the smallest arborizations of the bronchial tree. This wide penetration of the membrane is beautifully shown by Tull[3] in a recent case report with photographs, where a complete

cast of the bronchial tree was removed from a patient treated at the Sydenham Hospital in Baltimore. In conjunction with this clinical study, aerobic and anaerobic cultures were made from the membrane and purulent discharge removed from the larynx, trachea and bronchi. Many of the ordinary mouth organisms were found in these cultures, thus establishing that as the false membrane is formed by the Klebs-Loeffler bacillus, it carries with it contaminating aerobic and anaerobic organisms.

The protocols of 100 diphtheria deaths were also studied. These autopsies and protocols were made by Dr. Lawrence W. Smith, and I take this occasion to comment on the excellency of both. I quote the pulmonary findings of typical cases. "The trachea and bronchi contain a mucopurulent exudate and patches of membrane, especially the right bronchus, which contains a complete cast. A partial cast is also seen in the left bronchus. The smaller bronchioles throughout the lungs contain thick membranous casts."

In another case; "Throughout both lungs there is a patchy atelectasis and beginning bronchopneumonia. Many bronchioles contain membranous casts which extend down to the finest arborizations."

The autopsy diagnosis in one case was "necrotizing laryngotracheo-bronchitis," yet no lung abscess had formed. There was, however, a bilateral bronchopneumonia.

In an infant who died on the eleventh day of illness, "Examination of larynx and trachea reveals fragments of adherent membrane extending from just above the vocal cords as far as the tertiary bronchi. Most of the membrane was broken off and removed by suction but grayish, shaggy, dirty fragments are seen adherent throughout and blocking some of the smaller branches of the bronchi. This has resulted in a secondary pneumonic process."

In all autopsies reviewed, where pneumonia was a complication, it was described as a definite bronchopneumonia, a lobar pneumonia in various stages of hepatization, or a hypostatic pneumonia. Pneumonitis was not described as a pathological finding.

By mere coincidence, the one hundred and first protocol examined revealed a solitary lung abscess. This patient, however, had a tracheotomy wound which showed the same necrotizing infection as was found in the lung abscess. Could the presence of lung abscess found in this isolated case have been due to an embolus from a thrombophlebitis in the tracheotomy wound?

The finding of this case led to a review of all available tracheotomy cases on the diphtheria service. In all, 85 cases were found and in this small number 3 developed lung abscess. The only apparent difference between those tracheotomized and those intubated was the addition of an incision with its potentialities of thrombosis and embolus formation.

Permit me to introduce here a bit of experimental data found in an article by Lambert and Weeks.[4] They attempted to produce experimental lung abscess by injecting a potent mixture of bacteria into the ear veins of rabbits. None developed lung abscess except the last animal injected. This one, however, developed a thrombophlebitis of the injected vein. Could this abscess have been due to an embolus from the involved vein?

A special study of postoperative pulmonary complication is being made on the Fourth Surgical Division, Bellevue Hospital. This study has not progressed to a place justifying a detailed report. It can be stated, however, that pulmonary lesions of minor degree, where clinical manifestations are limited to a very slight disturbance such as a mild rise in pulse and temperature, with possibly a transient pain in the chest, or where the radiogram reveals a small area of consolidation, are astonishingly frequent. If these minor infarctions occur not infrequently and admittedly, the serious pulmonary thromboses are not infrequent, then it must follow that the intermediate sized infarctions, which produce tissue death, are also not

rare. These small infarctions, of necessity, form in the smaller calibered vessels which, of course, are found in the lung periphery. Given an infected embolus or even a fair sized sterile embolus which causes ischemia, resulting in tissue death, plus mouth organism contamination through the bronchus, lung abscess follows and cannot be distinguished from the bronchogenic type of solitary abscess. As only part of these abscesses would be expected to communicate with a bronchus, we naturally find the small, clinically insignificant, non-bronchial communicating abscess which heals in two or three weeks, as well as the fulminating putrid abscess.

If this theory is accepted, then the relative rarity of lung abscess is explained more easily since infarction itself is relatively rare and only a portion of the infarctions possess lung abscess potentialities.

It is an undisputed fact that lung abscess occasionally follows tonsillectomy and it is claimed that the aspiration theory readily explains its occurrence. Many observations have been made in support of this theory. Prophylactic measures have been suggested and adopted, such as local anesthesia, dependent head position, expert anesthetists who do not abolish the laryngeal reflex during the operation, and the use of a dependable vacuum suction machine. In spite of all these precautions, posttonsillectomy lung abscess is still far too common. Indeed, it occurs more frequently in the modern operation of tonsillectomy than in the days of tonsillotomy when few, if any, of these precautionary measures were practiced. In explanation of this fact, let us review the anatomy of the tonsillar region and its bearing on the operation of tonsillectomy versus tonsillotomy.

It will be recalled that the tonsil, lying between its pillars, is surrounded by the plexus tonsillaris and plexus pharyngus, which cover the outer walls of the pharynx and posttonsillar spaces. These venous channels receive blood from the tonsils, tonsillar pillars and pharyngeal wall and due to their anatomical situation, are exposed and always injured even in the gentlest technique of tonsillectomy. While in the operation of tonsillotomy, the protruding portion of the tonsil is guillotined without injury to the venous plexus. However, the same aspiration possibilities obtained in both procedures.

Given an injured plexus of veins, which drain through the descending pharyngeal plexus into the internal jugular and upward through the posterior palatine into the external jugular vein, it is not difficult to believe that infected emboli could be dislodged from veins thrombotic from trauma and infection, which are located in an area active in every attempt at speech and swallowing. Is it not more logical to believe that posttonsillectomy lung abscess is more often initiated by embolus than by aspiration, especially since it has been shown that 80 per cent of tonsillectomy patients aspirate pus, blood and corruption during the operation, yet lung abscess is so relatively infrequent? If Moore's[5] estimate of one lung abscess in 2500 cases of tonsillectomy is approximately correct, and if 80 per cent of those operated upon for tonsillectomy aspirate mouth contents into their bronchi, then only 1 in 2000 that have actually aspirated mouth contents into their bronchi develops lung abscess. By mathematical probabilities this is difficult to believe. And furthermore, bear in mind the relative ease with which lung abscess is experimentally produced by way of the blood stream, and the difficulty of producing it by way of the bronchi even when large potent doses have literally been rammed into the bronchi of non-immunized animals and held there with cotton plugs.

Most of the preventive measures in use today have been evolved from consideration of the aspiration theory. In the field of tonsillectomy alone, many articles have been written to promulgate preventive measures. These measures; viz., expert anesthetists, competent vacuum suction, oral sepsis with the removal of particulate matter from the teeth, ligation of bleeders, and posture during and immediately after

tonsillectomy are excellent and should be demanded. But, when in spite of their careful application and in spite of the numerous papers stating that "with our technique we have done 15,000 tonsillectomies without a case of lung abscess," the thoracic internist and surgeon continue to see an increasing number of lung abscesses, does it not connote that some other common etiological factor is active which the preventive measures have not eliminated?

The practical and clinical reasons for discussion of these two well known theories is to better focus our attention not alone on the necessity of using all known preventive measures, but to suggest the direction of research in evolving a better control of this serious complication. The writer believes that investigation along the line suggested by the embolic theory will be fruitful in preventing not only post-tonsillectomy lung abscess, but all post-operative lung abscesses, and possibly even those not connected with operative work.

Much work in this direction has been done and the latest sponsor, whose work is of particular value, is Bancroft[6] and his colleagues. In a recent interview he advanced the idea that just as we have patients with hypergastric acidity and others with hypogastric acidity, hyperthyroidism and hypothyroidism, so we have hyperbleeders and hyperclotters.

Since, as Bancroft has shown, there are individuals who have predisposed clotting factors, would this not be an explanation why thrombosis, embolism, infarction and lung abscess occur in one individual and not in another, both of whom have had the same operative technique and post-operative treatment?

Suggestions to prevent thrombosis:

1. Intensive research to unravel the clotting factor riddle;
2. Gentleness in handling or manipulating tissue;
3. Protection of exposed tissue from drying; i.e. cover exposed tissue with moist saline sponge;
4. Prevention of dehydration before, during and after operation;
5. Scrupulous asepsis, and in infected wounds, avoidance of physical trauma by retractors or of rough handling;
6. Avoidance of mass clamping and mass ligation of bleeders;
7. Careful approximation of tissue in closing the wound to produce speedy healing;
8. Avoid transfixion of tissue in infected or potentially infected wounds (tonsillectomy);
9. Avoidance of irritating chemicals on exposed tissue, such as pouring iodine or ether into a wound;
10. Avoidance of unnecessary speed, also unnecessary prolongation of the operation.

TREATMENT

In January, 1936, a comparatively large series of patients suffering from putrid lung abscess, all of whom had been operated within a period of six weeks from the inception of the abscess, was shown before this Society. I am in full accord with this type of treatment; it is a logical conclusion, which surgical practice must accept, that the sooner an abscess of the lung can safely be externally drained, the better the result. There will be less destruction of pulmonary tissue, less chance of metastasis to the healthy lung and convalescence necessarily will be shortened.

Admittedly, this treatment is not advocated for the non-fulminating pyogenic type of abscess which has been shown, both clinically and experimentally, to subside spontaneously. The treatment of watchful waiting in the latter type of abscess is very gratifying. This type, however, must not be confused with the fulminating putrid abscess.

Unfortunately, my experience in most cases has been to treat lung abscess not in its incipiency where early operation can be performed, but in the late stages where every other treatment has been tried and failed.

I know of no more difficult or discouraging task than to be asked to treat surgically these chronic abscesses that have existed for months or even years. To effect a cure, they essentially require radical surgery which necessarily carries a high mortality. Yet, at this stage, to continue medical treatment means 100 per cent mortality. Can surgery justifiably refuse treatment when it still holds a possibility of recovery or improvement?

Illustrative of this, I cite Case III.

Case III. C. C., admitted to Knickerbocker Hospital, gave a history of tonsillectomy followed by lung abscess, two years prior to admission. He had been treated by private physicians and in a number of public hospitals. He had a septic fever and profuse, malodorous expectoration. His latest and most pressing complication was an acute infectious enteritis. He was treated symptomatically until a differential diagnosis between tuberculosis and lung abscess could be established. When transferred to the surgical ward, he was essentially a very poor surgical risk.

Under local anesthesia a portion of rib was resected and the abscess was drained externally. The patient was relieved immediately of his profuse expectoration, but in ten days he added one more death to the surgical mortality of lung abscess. Here let me state a peculiar attitude taken by the laity, and by some physicians. If this patient had continued under medical care, his death would have been classified as "an act of God"; operated, it was classified, at least to some extent, as an act of the surgeon.

That a fair percentage of lung abscesses will recover by conservative treatment is readily admitted. It consists of bed rest, postural drainage, and such drugs as salvarsan, expectorants and those used for the relief of pain, none of which directly influence the course of the disease. Bacteriophage and vaccines have been recommended and used, but they have not been convincing in their results.

Of all the conservative armamenta, postural drainage is probably the most effective and the most abused. It is recommended for all types of pulmonary suppuration. It is of great value in chronic bronchiectasis, but is of value only in an occasional case of acute lung abscess, indeed many times it is distinctly harmful. Brunn[7] calls attention to this fact and states, "I am cognizant of three cases in which death resulted from its application." It has been noted by a number of observers and certainly one must agree that, "To apply this remedy to a very sick patient, cyanotic, with high fever, rapid pulse and a low vital capacity, is courting disaster."

Those who insist upon subjecting all patients suffering from this disease to a long period of non-operative treatment should assume the responsibility for these poor operative risks when they ultimately come to operation, and the surgeon is faced with the end pathology of the disease such as hemorrhage, marked fibrosis, pleural or secondary pulmonary abscess, or bronchiectasis, all of which require radical surgery for cure.

Pneumothorax although frequently used as a treatment of lung abscess, is seldom justifiable. In cases of severe hemorrhage, it may be helpful and warranted, but applied as a regular form of treatment, it must be condemned. Drainage is the essential factor in the healing of lung abscess, and pneumothorax may help or hinder the mechanics of drainage. The main objection, however, is when rupture into the pleural cavity takes place. If this occurs and the lung is normally expanded, there is more probability of forming an encapsulated pleural abscess. With pneumothorax, however, general pleural involvement occurs, resulting in severe shock, rapid intoxication and high mortality.

It is tragic to observe the speed with which a comparatively well patient may become desperately ill when a lung abscess ruptures into an artificial pneumothorax. Illustrative of this, I cite Case IV.

Case IV. E. B., had an abscess of the right upper lobe. Complete collapse of the lung, except that portion overlying the abscess which was adherent to the chest wall, was obtained by pneumothorax. Her temperature had been

practically normal for weeks until one morning at four o'clock she was awakened by a severe pain in her right chest. The abscess had ruptured, causing a soiling of the clean pleural cavity. The temperature within one hour reached 105°F. The patient was dyspneic, her heart action was very rapid, and she was a poor surgical risk. A simple thoracotomy was done with the patient propped in a sitting position, the only position that did not increase her dyspnea. Her recovery from the putrid empyema was slow and hazardous. Her abscess disappeared, leaving in its place a bronchiectasis in the involved lobe. This patient recovered but the mortality of such accidents is essentially very high.

Lobectomy, although a radical procedure' becomes the operation of choice in certain types of cases. It is indicated in multiple small abscesses limited to one lobe, or where hemorrhage is the urgent symptom. It is also indicated in longstanding cases where the abscess becomes a part and parcel of a complicating bronchiectasis, and in the rare case that develops a central or hilar abscess that does not heal spontaneously.

By preference lobectomy should be an actual surgical removal. If, however, circumstances will not permit such a procedure, a "cautery lobectomy" may be performed. The latter is an uncertain, messy type of surgery and fraught with many pitfalls and dangers. Delayed postoperative hemorrhage not infrequently occurs and usually it is not suitable for children as their nervous fortitude will not withstand the multiple attacks.

Case v. Patient L. W., was admitted to Bellevue Hospital with a central abscess just lateral to the hilum of the right lower lobe. She had been treated by all the conservative means, but frequent and alarming hemorrhage persisted. She was operated for lobectomy. No adhesions were found attaching the lung to the parietal pleura. A one stage lobectomy was performed, which resulted in a cure. On examination of the specimen, a vessel was found crossing the abscess cavity.

A second case that I now believe should have had a lobectomy, was treated by unroofing the abscess cavity through a large avascular area of the diseased lobe. The cavity was packed and no further bleeding took place, but the patient ultimately died of a metastatic brain abscess.

Phrenic. Phrenic interruption, according to the literature, has a place in a few selected cases. We have not had an occasion to use it in the treatment of lung abscess. Suffice it to say that it does not seem to be a logical procedure for the treatment of this disease.

Thoracoplasty. Selective thoracoplasty as an aid in healing a drained abscess cavity, which does not spontaneously close, is of distinct value. But thoracoplasty as a primary operation is not a logical procedure because it does not provide the two requisites for the healing of a lung abscess; viz., drainage and aeration.

Two statements are quoted from differing writers on this subject; one remarks, "Putrid lung abscess is potentially a surgical lesion from the onset. There is no proved method of definitely influencing the course other than by open operation." The other states, "Let them alone when they are acute. I think we would have far better results in the treatment of lung abscess if we let more of them alone."

These two statements might readily have been made concerning the treatment of appendicitis in the early nineties. In fact, my earliest memory of reading a medical article was entitled "Medical Treatment of Acute Appendicitis" by Dr. Green. In this paper he advocated letting these cases alone in the acute stage. One cannot help but draw an analogy. As long as expectant treatment was the method of choice, the mortality of appendicitis continued high regardless of the fact that a certain percentage recovered, or at least did not die. This caused physicians to hope that every case would fall within this group.

When surgeons developed a satisfactory technique to manage acute appendicitis, gradually operative measures forced its recognition. So, too, will the improved technique of operative drainage of lung

abscess force its recognition and bring surgical intervention in the early stages, i.e., within the first four to six weeks. Then, and only then, will patients be spared the choice of chronic invalidism and untimely death, or the acceptance of radical surgery with its higher mortality.

CASE REPORTS

A large group of patients, who had been operated on for lung abscess within a period of six weeks of inception, was presented before this Society in January of this year. As a contrast, I have chosen to present a group of patients operated on very much later, that is, anywhere from six months to two years after the inception of their disease. These patients have come from Bellevue and Knickerbocker Hospitals.

Case I. E. B., a girl of eleven years, was admitted to the medical service of Bellevue Hospital in 1929, with physical signs and *x*-ray films showing what proved to be a pneumonitis of her right upper lobe. She was treated by orthodox medical procedure but the process did not recede; on the contrary, it was progressive. In the face of the fact that this was an apical lesion, which makes a surgical approach difficult, a pneumothorax was tried. This served to aid in making a more accurate localizing diagnosis and also as a therapeutic measure. Improvement followed this treatment, her temperature became normal, and it seemed a real therapeutic success. However, about six weeks after the induction of the pneumothorax, she was awakened from a sound sleep at 4 A.M. by a severe pain in her right chest. The abscess had ruptured into her clean pleural cavity, causing a generalized putrid empyema. It was tragic to observe how quickly she became a desperately sick child. Her temperature rose to 105°F. within three hours. Dyspnea was very distressing, and the pulse rate rapid. An operation of any magnitude was impossible, so a simple intercostal drainage was done under local anesthesia.

After a stormy course, she recovered. Recently an upper lobe bronchogram was made to determine what, if any, lung pathology remained. She has a moderately severe bronchiectasis of her upper lobe, although she has practically no clinical symptoms from this condition. It will be interesting to see if, at her age, she will outgrow this bronchial dilatation.

Case II. This girl E. B., aged ten years, came to Bellevue Hospital four years ago with a lung abscess which she had had for six months. She was expectorating very thick, foul pus and an *x*-ray film revealed a fluid level. We questioned whether we were dealing with a pyopneumothorax or a lung abscess, or both. To aid in making a differential diagnosis, a bronchogram revealed a well developed bronchiectasis, apparently the cause of her expectoration.

At operation, under local anesthesia, the lung abscess was drained. Later, a retrograde bronchogram showed a huge cavity in the lower lobe. A portion of the lipiodol was aspirated in the contralateral lung, filling the bronchi even to their finest arborizations. Certainly the lung abscess pus must have done likewise on many occasions, yet no bronchogenic spread took place. This is usually the case and, I believe, it is an argument against the aspiration theory as the genesis of lung abscess.

The patient was discharged with her wound healed. However, in the follow-up clinic it was apparent that she was not well, although she was active and had no expectoration. At the end of her fourth postoperative year she was readmitted for a six week's observation. She was found to be underweight, undersized and undernourished. She had no expectoration, but did have a persistent temperature of 101°F. A bronchogram showed that the bronchiectasis had advanced. There was fibrosis of the left lower lobe with many large bronchiectatic abscesses. A one-stage lobectomy was done, which, of course, was for the bronchiectasis. However, since this was a mere complication of the lung abscess, I am including this child in the group of lung abscess patients.

The pathologist reported the removed lobe to be fibrosed, containing no air and no evidence of the original abscess cavity. The patient is now well and has a normal temperature.

Case III.* L. U., a girl of eight years, had a lung abscess following tonsillectomy, and her chief symptom was hemoptysis. She had very severe hemorrhages which persisted despite all possible medical care. A one-stage lobectomy was done, and when the excised lobe was

* This case was reported in detail in the *Am. Jour. Surg.*, 34: 83–89 (Oct.) 1936.

injected it showed what I interpreted as a centrally located lung abscess, a little closer to the mediastinum perhaps, but it could hardly be called a peripheral abscess. Strangely enough, there were no adhesions whatsoever to the pleura.

Case iv. A twenty-seven year old woman, J. M., was admitted to Knickerbocker Hospital in 1933, remaining on the medical service for three months after which she was discharged as cured. In two weeks her symptoms recurred, and in three or four days she ruptured an abscess into a bronchus, expectorating putrid material. She returned to the hospital as soon as she was able to do so and we decided to treat her surgically. It was difficult to make a localizing diagnosis but we believed the abscess was at the level of the sixth rib in the posterior axillary line. Accordingly, her chest was opened at this point and we found a thin pleura with the lung moving under it. Knowing that we had practically exhausted our ability to localize the abscess roentgenographically, I deliberately made a hole in the pleura and explored it with my finger. The abscess was readily localized at the level of the fifth rib under the angle of the scapula. The wound was closed but eight days later we resected 1½ inches of scapula, removed the fifth rib, and aspiration revealed the abscess which was then unroofed and packed. She made an uneventful recovery. This was done three years ago, and she has had no further symptoms.

Case v. Mr. V. Z., aged forty-seven years, came to Knickerbocker Hospital complaining of bloody expectoration. He had had several rather severe hemorrhages. The *x*-ray film showed consolidation of the left lower lobe, which may have been due to atelectasis, fibrosis or a lung tumor. The pleuras were adherent. We did not believe a bronchogram would be of much diagnostic assistance, but he was bronchoscoped and an obstruction was believed to be seen in the lobe bronchus, such as might be caused by a tumor pressing on the bronchus. A biopsy proved to be normal bronchial mucosa. With the story of expectorating a good deal of blood and associated loss of weight in a man of forty-seven years, we suspected that we were dealing with a new growth, although we were unable to make a positive diagnosis. The question arose as to whether a lobectomy was warranted without a more definite diagnosis. We decided we were justified, our attitude being that if we waited until a positive diagnosis could be made it would be too late for surgical intervention.

Upon exploration we found that the left upper lobe was entirely normal, the lower lobe was fibrosed and contained multiple abscesses, but no new growth. A one-stage lobectomy was done. The patient made a very uneventful convalescence in spite of a good deal of infection in his wound due to Welch bacillus.

SUMMARY

1. A discussion of the aspiration and embolic theories of lung abscess is presented.

2. Case histories are cited to substantiate conclusions that pulmonary embolism is frequently followed by typical lung abscess.

3. In a study of 100 diphtheria death protocols, false membrane, with its contaminating mouth organisms, was found in the minute arborizations of the bronchial tree, but no lung abscess complication was found.

4. When operation (tracheotomy) was performed on bronchial diphtheria patients, abscess was found in 3.5 per cent of the cases.

5. Many preventive measures have been devised and are used to prevent aspiration during tonsillectomy, yet far too many patients develop lung abscess. Unquestionably, lung abscess is a more frequent complication of modern tonsillectomy than the older operation of tonsillotomy.

6. Earlier surgical drainage is urged in fulminating lung abscess.

7. The use of drugs and vaccines is of no value in the treatment of lung abscess.

8. Pneumothorax should not be used as a treatment of lung abscess. It may be used as a localizing diagnostic measure.

9. Postural drainage must be used cautiously in acutely ill patients.

10. If surgical treatment is postponed until a chronic stage has been reached, more radical surgery, with its higher mortality, is required to effect a cure.

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BANTI'S DISEASE WITH GASTRORRHAGIAS AND THROMBOPHLEBITIS*

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THE frequent pre- and postoperative hemorrhages from the gastrointestinal track in splenic anemia present a serious problem in the treatment of Banti's splenomegaly. Several recent case reports,[1–5] especially that of Pemberton's,[6] demonstrate the gravity of this gastrorrhagic tendency. The amount of blood lost varies from a few drams to one quart or more at each bleeding. The onset of hemorrhages occurs without any prodromal signs and takes place in the second and third stage of Banti's disease. The youngest patient with gastrorrhagia was eighteen years[7] and the oldest fifty-eight years.[3]

The causative factors may be divided into two groups: (1) mechanical and (2) physiopathological.

The enlargement of the spleen, by its size and weight, may compress and distort the lineal vein, thus impeding its circulation. Such a compression may also establish secondary traumatic changes in the wall of blood vessels leading to aseptic thrombophlebitis of the splenoportal veins.

That the size of the spleen, per se, is not a sufficient factor in obstructing the venous flow from the spleen is demonstrated by splenomegalies of other diseases without gastric hemorrhages such as Gaucher's.[8]

The tendency to gastrorrhagias is due to a disturbance of the hemolytopoietic system.[9] It is probable that this disturbance embraces the whole reticulo-endothelial system as a toxicohormonal dyscrasia evidencing itself by predilection in the spleen and splenoportal region. Tripoli[10] states that the etiology of splenic anemia may be uniform, being produced by multiple, partly known causes and may vary in symptomatology, depending on whether it affects different organs in succession or simultaneously. Epinger and Brandelberg[11] deny a distinct entity to Banti's disease, arguing that histopathology of splenic anemia is almost identical with lesions found in certain cases of cirrhosis of the liver, tuberculosis, malaria and syphilis.

There are no positive diagnostic criteria from either a clinical or a laboratory point of view. The cytology of Banti's disease indicates only a secondary anemia with diminished red blood cell count and hemoglobin in proportion, and with the white blood count also diminished correspondingly.

These cases with marked leucopenia and increased urobilin and urobilinogen, indicating progressive destruction of blood elements, have a bad prognosis after splenectomy.[12] Similarly after marked gastrorrhagias with depressed bone marrow response, as indicated by the absence of nucleated red blood cells in the circulation, splenectomy is futile.[5] The only favorable sign is increase of cholesterol, such a case being reported by Hanrahan[12] twenty-five years after splenectomy.

Rosenthal[13] distinguished two types of splenic anemia cases: (1) those with thrombocytopenia and favorable prognosis after splenectomy and (2) those with thrombocytohemia with marked tendency to postoperative thrombosis in the splenoportal region. Consequently Rosenthal[13] and Chevalier[14] caution against splenectomy in the presence of thrombocytohemia.

The bleeding and clotting time, fragility of the red blood cells and tourniquet tests do not yield any positive corroborative evidence in splenic anemia. In suspected

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cirrhosis of the liver, secondary to splenomegaly, bromsulphalein and galactose tests may reveal the extent of liver damage. One of the more useful differential tests is the action of adrenalin on the enlarged spleen. Thus in Banti's disease and infectious splenomegaly the spleen does not contract after an injection[15] of adrenalin but does contract in splenoportal thrombophlebitis.[16]

Thus the physiopathological factors that lead to gastrorrhagias have their origin in the functional disturbance of the reticuloendothelial system and express themselves in the histopathology of the spleen and splenoportal region.

The great amount of blood demanded by the enlargement of the spleen is impeded in its course thus raising the intravenous pressure in splenoportal and gastroesophageal region. The splenic veins carry about one-fifth of the total portal circulation.[17] The blood vessels most involved are: splenic veins, left gastroepiploic and esophageal veins with the short gastric branches of the lienal artery and the left gastroepiploic artery. Pathologically there is thinning and stretching of the vessel wall with microscopical changes of all the coats[16] forming marked gastroesophageal varices. Their presence can readily be ascertained by esophagoscopic examination.[18] Walters[19] suggests that properly made injection of sclerosing solution into esophageal varices may prevent otherwise inevitable gastric hemorrhages, but the danger of precipitating gastric emboli must be considered. The presence of marked gastroesophageal varices may be suspected by the appearance of abdominal rete; this can readily be discerned by the employment of infra-red photography.

Whether gastrorrhagias are due primarily to over-distention of the gastroesophageal plexus or are dependent on some other physiopathological factors is not determined. Frugoni states that in splenomegaly with gastrorrhagias there must exist thrombophlebitis of the portal or splenic vein. Corroborative cases are described by Epstein,[16] Gamma,[5] Gasparini[15] and Lichtenstein.[7] Rosenthal's thrombocytohemic cases would also belong to this group. However, Pons[20] quotes a case of repeated hematemesis with splenomegaly but at postmortem no sign of thrombophlebitis of the splenoportal region was found. This case was only twenty-seven years of age.

It appears that thrombophlebitis is a part of Banti's syndrome, its presence depending on the stage of the disease and severity of the hemolytopoietic dysfunction as it affects the spleen. The severest venous lesions are found near the splenic hilum[21] and diminish in severity as they approach the portal trunk. Epstein[16] does not believe with French authors that a primary thrombophlebitis of the splenic vein exists. It appears that a stasis of the splenic vein plus the occurrence of thrombocytohemia, plus the traumatic and toxic injury to the intima will predispose to thrombophlebitis. This occurs in the third stage of Banti's disease and in about 30 per cent of cases it is associated with ascites and cirrhosis of the liver.

Clinically the presence of gastrorrhagia and the absence of ascites practically excludes Banti's disease.

Another significant factor in this connection is the presence of perisplenitis with multiple adhesions of the spleen to its surrounding viscera and adhesions of the omentum to the anterior abdominal wall. Such adhesions indicate the presence of congestion in the splenoportal region and an attempt to establish collateral circulation.

In one of the recent cases observed the incision of the abdominal wall presented an area of tremendous oozing and the entire omentum was adherent to the anterior abdominal wall. Loeffler[22] in his experiments on rabbits demonstrated the establishment of collateral circulation to the suprarenals and also to the greater curvature of the stomach.

The results of splenectomy are satisfactory in this sense that noxious substances causing splenomegaly and anemia

are removed; the general condition of a patient is improved and in many cases life is prolonged from two to twenty-five years. Splenectomy, however, does not prevent recurrence of gastrorrhagias in some cases.[5,7,20,23] The factors responsible for the improvement in some cases briefly stated are, relief of pressure in the splenoportal region and removal of the thrombolytic factors.[13] As a corollary to this improvement it is observed that there is a remarkable power of the liver to regenerate with disappearance of the cirrhosis and reduction of pressure in the splenoportal region.

It appears that when cirrhosis of the liver secondary to splenic anemia is advanced far beyond possibility of repair, passive congestion in the gastroesophageal varices will persist after splenectomy unless efficient collateral circulation is established. Pemberton's method of step-like omentopexy[6] solves this problem to a certain degree.

A more formidable procedure and one that would give complete relief of splenoportal obstruction would be a direct anastomosis of the distal end of the splenic vein to the inferior vena cava or azygos vein on the left side. But above all to obviate any possibility of gastrorrhagias an early recognition of splenic anemia and early splenectomy must be performed before the onset of cirrhosis of the liver and passive congestion of the gastroesophageal veins take place.

CONCLUSIONS

1. A review of literature on splenic anemia associated with gastrorrhagias is presented.

2. Causative factors in the production of gastroesophageal varices and thrombophlebitis are reviewed and discussed.

3. Methods of preventing gastrorrhagias are suggested.

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OPERATIONS FOR CANCER OF RECTUM

EXPERIENCES AT THE UNIVERSITY OF CALIFORNIA HOSPITAL

MONTAGUE S. WOOLF, M.D.

SAN FRANCISCO

ACCORDING to Hayden,[1] who has summarized the history of procedures for cancer of the rectum and whose account I have used freely below, Lisfranc was the first to amputate the rectum and to report 9 cases from 1828–1830, but operated only for growths which were very low. For these he made an encircling incision around the anus and dissected close to the rectal wall and then cut across the bowel. In 1873, Verneuil and Kocher removed the coccyx. Kocher also sutured the anus to prevent sepsis. Sir James Paget apparently planned the first excision in England in 1878. Harrison Cripps in the same country in 1884 published 3 cases of perineal excision. One survived several years. Kraske in 1885 removed part of the sacrum but the sphincter was preserved and the bowel above the resected portion pulled into the sacral wound. For over twenty years such an operation was used almost universally. Recurrence prior to 1884 was unlimited. By 1910, W. Ernest Miles of London was definitely committed, owing to his idea of the wide spread of invasion in several directions which attends a growth in the rectum, to the idea of an abdominoperineal operation with a colostomy. In this operation, which he performs at the present time exactly as he described it then, the bowel, the pelvic mesocolon, the perirectal glands and fat, the ischiorectal tissues and the sphincters were removed. This operation, done in one stage, leaving the patient with a permanent colostomy, was in conception and even in practice of magnificent proportions. It is of all procedures the one most likely to prevent recurrences, but it has been one, too, to which the patient most often succumbed.

Such a procedure with, at first a grave mortality of 41 per cent even in Miles' hands, induced W. J. Mayo in 1912 to develop a technique which divided such an operation into two stages. He made an end colostomy and inverted the end of the distal portion containing the growth until a later date. His mortality was 13.3 per cent in 30 cases and as compared to 35 per cent when he used Miles' single stage method the former figure was a great improvement. The trouble with this two stage operation was that the blood supply of the distal loop was cut off so that it became gangrenous and septic absorption from this often killed the patient before the second part was done. Therefore, Coffey devised his quarantine drainage of the infraperitoneal space in which the dead segment of bowel lay and reported a mortality of only 5 per cent. No one else has been able to approach so low a figure for such an abdominoperineal excision and it seems unlikely it will be repeated, unless candidates for it are carefully selected.

Other abdominoperineal operations have been devised; in 1915, D. Jones of Boston mobilized the sigmoid, preserved an adequate blood supply to the sigmoid and rectum, reconstructed the pelvic peritoneum to a higher level, depressing the sigmoid below it. Then he constructed a loop colostomy higher up, later removing the rectum and sigmoid from the perineum. This meant that in his second stage he was able to excise a good length of sigmoid with the rectum so that a growth could be removed higher up than in the Mummery type of procedure which it resembles. His average mortality is 22 per cent.

[1] HAYDEN, E. P. *New Eng. Jour. Med.*, 206. (May) 1932.

Lockhart-Mummery opens the abdomen, performs a loop colostomy and removes the rectum from the perineum with as wide a dissection as possible. The coccyx is always excised. His mortality is 3 per cent in private cases and 14 per cent in charity cases. He obtained 54.8 per cent five year cures.

Lahey divides the sigmoid, brings the upper end out in the iliac region and implants the distal opening in the exploratory wound. Later the distal opening is closed, the abdomen again entered and the distal loop depressed and removed at the same operation through the perineum. The lower segment can be cleaned this way in the interval but the abdomen is entered twice, adhesions have formed possibly and the field is liable to be contaminated. It is useful in obstructed cases but one would think, if the obstruction is at all serious, a simpler and more rapid decompression of the bowel might be more appropriate.

More recently, Rankin and Gabriel have published reports of a reverse type, a perineo-abdominal operation of similar characteristics. A loop colostomy is performed. At a second procedure the rectum is dissected up as in the Mummery procedure but on opening the peritoneum the bowel with its growth is pushed into the abdominal cavity from which it is removed by an abdominal incision. This operation allows the removal of a tumor situated high up, if it cannot be done through the perineum alone. But not infrequently the rectum may be opened accidently and the field contaminated. The abdomen has been entered also late in the operation and anesthetic difficulties may be encountered especially if a thecal analgesia with or without gas and oxygen has been used. Two major abdominal incisions have been made.

In discussing the question of what course of treatment should be adopted for any surgical condition, it will be found that the most desirable operation is the one that can be used most extensively under varying conditions; a minority procedure on the other hand, in time disappears but operations must be done finally in all areas. There is also a philosophy in surgery which does not have much to do with ultimate statistics of rates of immediate and remote mortality but which has its importance in selecting the operation. It must be remembered that the surgeon is likely to have 2 patients after an operation which entails a colostomy, the one being the patient, the other the family of the patient. There are tribulations connected with the possession of a colostomy which pervades the house of the one who has it. But it is equally certain that a colostomy has to be advised if the best hopes of the patient, which means life, are to be realized. The evolution of the operations for cancer of the rectum easily demonstrate this. Again, these facts are clear: a successful colostomy alone will prolong life; a serious primary mortality is not compatible with the hopes of the relatives of a patient who expend their means trusting to keep him alive as long as possible, for the expense may well approximate the same figure whether the patient lives or dies. Having these considerations in mind, we will return to certain figures regarding the operative mortality and rates of recurrence.

TABLE 1

TOTAL NUMBER OF CASES OF CANCER OF THE RECTUM—86

Operable cases	46	53.5 per cent
Inoperable cases	40	
Females	26	
Males	60	

AGE INCIDENCE

Years	No. Cases
10–20	1
20–30	3
30–40	11
40–50	13
50–60	28
60–70	24
70–80	6

Operative Mortality Analysed:

Exploratory laporatomy and colostomy	6 deaths
Resection	8 deaths
Total operative mortality	16.2 per cent
Excluding deaths from colostomy	9.3 per cent

The total number of cases of cancer of the rectum at the University of California since 1915 have been 86. Varying types of

operations have been done from the exact procedure of Kraske to that of Miles. The operations were performed by many general surgeons but by many fewer since 1926.

Table I shows certain facts, namely, that 53.5 per cent were operable and that there were more than double the number of males than females. Operative mortality was 16.2 per cent, but if one ignores those dying from the colostomy alone, done very often for emergency and palliative reasons and carries a high death rate, then there were 8 deaths in 86 cases, or 9.3 per cent.

TABLE II

POSTOPERATIVE FOLLOW-UP

38 cases who have had follow-up (2 cases not followed) following resection of growth

19 cases still alive following resection; (19 cases dead since resection)

DURATION OF LIFE

Since Operation		After Operation
Years	No. Cases	No. Cases
Under 1	6	4
1	3	7
2	2	3
3	1	
4	2	
5	0	1
6	0	1
7	2	1
8	1	
9	..	1
10	..	1
11		
12		
13		
14	2	
Total	19 deaths since resection	19 survivals

Number of 5 year cures still alive..... 5
Number of 5 year cures dead..... 5
Total 5 year survivals..... 10
Percentage 5 year survivals in resected operable (46) cases..... 21.7 per cent

Table II shows the fate of 38 followed cases after resection; 19 were still alive and since, according to authorities[1] colostomy alone does not prolong life two years, we may consider that of these 19 cases 10, or 53 per cent, were recipients of prolonged life although in some the length of ultimate survival is not yet known. Nineteen followed since resection died but these also were benefited to the extent of 8, or 42 per cent, having lived over two years. If a five year survival is a basis of estimating the value of resection, then it will be seen that of 46 resected cases, there were 21.7 per cent five year survivals.

Since 1926 at the University of California there has been an active proctologic department from which most of the resections of the rectum have been handled, in nine years there having been 38 operable cases. In these there had been but one death, a mortality of 2.6 per cent, as Table III shows.

TABLE III

38 operable cases who had resections
1 postoperative mortality = 2.6 per cent
14 cases dead since resection
23 cases still alive since resection

ANALYSIS OF RESULTS OF OPERATIVE PROCEDURES

Operations	Number of Cases	Operative Mortality	Five Year Survivals
Lahey	1		
Coffey (2 stage)	5	..	2
Rankin (2 stage)	1	1	
Miles	2		
Lockhart-Mummery	27	..	4
Local excision	2	..	1

About two-thirds of these have had the perineal resection of Mummery, 18.4 per cent having survived more than five years. In addition, Table III shows that of those dead of recurrence 3 survived over five years; in other words, so far there has been a 26 per cent five year survival from the Mummery operation.

These figures show nothing more than that in a modest way we, too, have improved markedly the primary mortality and to a less extent lowered the recurrence rate after operations for cancer of the rectum.

Our experience with the Mummery operation has been eminently successful as

[1] Ministry of Health, Report on Cancer of the Rectum, London, 1927.

far as the primary mortality is concerned; one death in 38 resections. By removing the lower portion of the sacrum in addition to the coccyx, we have always been able to remove a growth in the rectum or at the rectosigmoid junction. No selection of cases has been made; our figures include even growths that have perforated the outer coat of the rectum and in one case invaded the prostate. However, if a patient is of an age and build to indicate that a major abdominal operation would not entail grievous risks, we perform an abdominoperineal operation, preferably in one stage.

Earlier I mentioned many operations devised to circumvent a high primary mortality and a large recurrence rate. They all have been very helpful and each has been successful in the hands of their originators, but I believe one has to confine oneself to advising the simplest of procedures for general widespread use.

It is impossible for the outlying surgeon to select definitely a major procedure from many variations by the method of trial and error. Therefore, it is my suggestion that the perineal operation of Mummery and the one stage abdominoperineal operation of Miles be adopted as the two important procedures for use in removing the cancerous rectum. The second part of the latter can not have a great effect on operative recovery. The procedure of a few minutes removes the mobilized bowel. A patient does not die from this but from the effects of the much greater intra-abdominal manipulation.

SUMMARY

The first amputation of the rectum was performed by Lisfranc in 1828. Up to 1884, even with improved operations recurrence was universal. At the present time if patients are subjected to a suitable operation for a removal of a growth the primary mortality need not be more than about 3 per cent to 10 per cent, according to the type of operation. The two operations being used with increasing success at the University of California Hospital are that of posterior resection preceded by a colostomy and the abdominoperineal operation in one stage. Experience plays a great part both in judgment and technique. This is shown by the reduction of mortality after 1926 when the operations were performed mainly by a single person. This figure has fallen from 16.2 per cent to 2.6 per cent. The rate of recurrence has not been reduced nearly as much, the respective five year survival figures being 21.7 per cent and 26 per cent, but we are not performing as yet the maximum number of one stage abdominoperineal operations.

CARCINOMA OF BLADDER*

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CHICAGO, ILL.

A DISCUSSION of tumors of the bladder limits itself for practical purposes to one of carcinoma of the bladder. Epithelial tumors comprise almost all the new growths which require diagnosis and treatment; and today they are all considered potentially malignant. Most will show, if left untreated, both clinical and histological evidences of malignancy.

The subject of bladder carcinoma has been the focus of interest of many during the last decade and with the exception of the type of therapy used, there is quite general agreement.

In this paper, based on a series of approximately 200 thoroughly controlled cases observed at the Edward Hines Memorial and the Michael Reese Hospitals, only the essential facts can be emphasized. Barringer aptly says, "controversial points, and there are many concerning bladder cancer, cannot in the least be settled by publishing statistics. These latter can only give a vague clew as to the approach of anyone surgeon in any given class of cases."

The etiological factors involved in the vast majority of bladder carcinomas are entirely unknown. All are familiar with the frequency of its occurrence in employees in and about anilin factories. Recent work from Wilmington, Delaware, the site of the huge DuPont Dye Works, presents the following interesting data. In 1601 cystoscopies since 1931, of which 428 were reexaminations, 70 bladder tumors were discovered, which occurred in men who worked with these chemicals for an average of eleven years.

The years between fifty-five and sixty represent the peak age for bladder cancer and approximately 62 per cent occur between fifty and seventy years. The disease affects four times as many men as women. Most bladder carcinomas are located on the trigonum, on the lateral walls and in the neighborhood of the bladder neck. Approximately 65 per cent occur singly, of which one-half are of the higher grades of malignancy, while 35 per cent appear as multiple tumors of which only one-third are grouped as grades III and IV.

The symptoms of bladder cancer are primarily hematuria and pain, which unfortunately may appear for the first time when the disease is relatively far advanced. Blood in the urine is of great significance, being the initial symptom in 63 per cent in this series, and its origin must immediately be determined. Hematuria may be intermittent and recur only at intervals of weeks or months, but its appearance must be considered of serious import, until it is proved otherwise after complete study of the entire genitourinary tract. Pain may be a prominent symptom and also the first noted in tumors of the infiltrating type, but may be absent entirely in papillary neoplasms. Loss of weight, anemia, general debility and the usual signs of malignant disease appear later, after the opportunity of affording relief with a reasonable chance for cure has long passed.

The pain of cancer of the bladder is not characteristic; it may occur only when it is distended or at the end of micturition. Obstructive symptoms may be evident when an infiltrating tumor involves the vesical neck or a papillary new growth is located in its vicinity.

The direct diagnosis of bladder carcinoma is relatively simple and only the cystoscope will reveal the new growth and permit of an accurate estimate of its size, location and general character, whether

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pedunculated, sessile or infiltrating. Occasionally the introduction of a cystoscope may be difficult, impossible or inadvisable on account of excessive bleeding or urethral obstruction. Under such circumstances cystography is a valuable aid and will give material information by showing evidence of encroachment on the bladder lumen by papillary neoplasms, or distortion of the contour of the bladder wall by infiltrating tumors. Some urologists prefer pneumocystography to filling the bladder with contrast fluids as 12.5 per cent sodium iodide or 25 per cent sodium bromide. Not infrequently excretion urography has been of great diagnostic value in disclosing unsuspected malignancies of the bladder.

Fig. 1. Carcinoma of the urinary bladder, grade I. Uniformity of cells which arise from the stalks. Note the small vessels in the stalk. The tumor cells are particularly alike as far as staining quality and size are concerned. Papilloma of the urinary bladder.

Fig. 2. Carcinoma, grade II. The tumor cells close to the vessels are slightly different from those seen in the periphery of the tumor. The stalk is not well defined. Moderate variations in staining quality. A few mitotic figures are seen surrounding the vessels in the center of the picture.

In differential diagnosis, all other sources of blood in the urine must be considered, especially that which may arise from the kidney, ureters, prostate and urethra. Traumatic hematuria, as well as bleeding from hemorrhagic nephritis and the blood dyscrasias, must also be differentiated. Lesions of the bladder which should be mentioned are connective tissue tumors, which are very rare. Tumors and inflammations of adjacent organs, bony structures and vesical calculus must be considered. Perforation of an inflamed diverticulum of the bowel may closely simulate tumor of the bladder; osteomyelitis and sarcoma of the pubis may have to be excluded. Syphilis and certain rare types of cystitis also must be considered.

BIOPSY

Biopsy of bladder tumors is valuable and should be made, though we have not found it very reliable in the accurate grading of malignancy. Too often there is a great variation in the histology of sec-

tions taken from the surface of a papillary neoplasm and those taken from the base. Therefore, we are not in full agreement with some pathologists who state that an accurate determination is possible from biopsy material. We believe that an estimate of relative malignancy can be made but that strict grading of biopsy material is impossible.

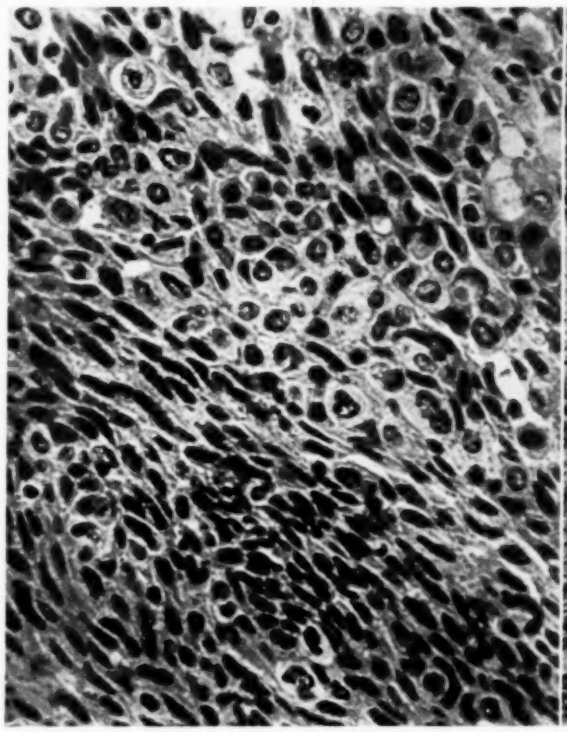

Fig. 3. Carcinoma, grade III. The variations in size, shape and staining quality are marked, some tumor cells being spindle shaped and others round. Distinct anaplasia is noted. Several mitotic figures are seen in the field (stalk is not present, sessile tumor).

Fig. 4. Carcinoma, grade IV, diffusely infiltrating cancer of the urinary bladder. Note the marked variation in size, shape and staining quality and the tendency of the tumor cells to assume the squamous.

PATHOLOGY

The pathology of bladder tumors presents variations and gradations from apparently benign papillomas with exquisite cell differentiation to very rapidly growing anaplastic carcinomas with either little or no cytological differentiation and presenting extreme variation in the nuclei, in staining qualities and many mitotic figures.

In patients affected with so-called anilin tumors which are probably due to certain chemical intermediates, particularly alpha and beta naphthylamine and benzidine, Gay has reported the occurrence of vascular changes, probably involving the entire bladder mucosa, but accentuated in the trigone, as the very earliest visible lesion. Thereafter epithelial proliferation occurs first in the basal and later in the superficial layers, with eventual appearance of papillary or sessile new growths.

As the histology of so-called anilin cancers is indistinguishable from that of the vastly larger group of tumors whose etiology and pathogenesis is unknown, it is possible that these mentioned changes may also be found in tumors of spontaneous origin. There are several facts which support this theory: (1) recurrences most often appear in the neighborhood of or at a distance from the original new growth rather than at the exact site of the primary neoplasm; (2) certain infiltrating carcinomas may involve the entire bladder wall, and even extravesicular structures,

when the cystoscopic appearance is that of a much smaller tumor.

A long discussion of the various gradings of bladder tumors is not offered, other than to briefly outline our classification, which is subject to change as new developments arise.

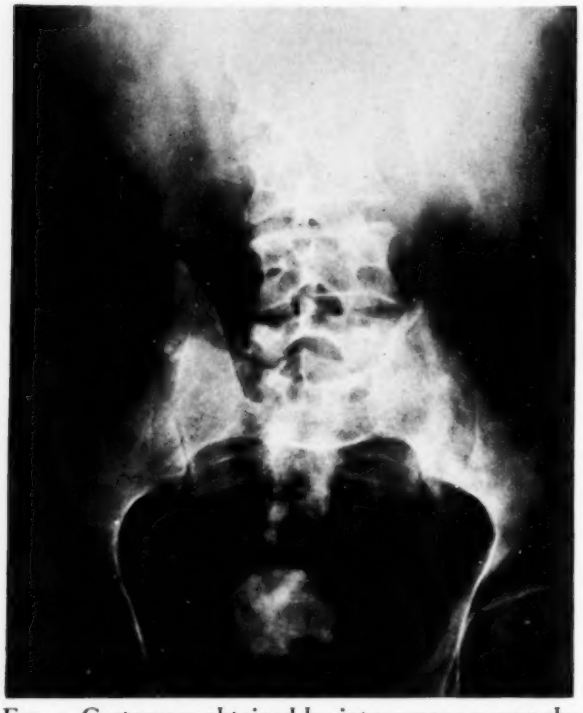

Fig. 5. Cystogram obtained by intravenous urography showing filling defect caused by carcinoma of the bladder.

GROUP I

This group includes small pedunculated neoplasms with thin pedicles, presenting definite wavy translucent or semitranslucent fronds, arising from normal appearing bladder mucosa and frequently are called benign papillomas.

GROUP II

This type includes usually somewhat larger pedunculated papillary new growths often having a thicker pedicle, in which some of the fronds have lost their semitranslucence as a result of epithelial thickening and fusion of some of the papilla. This type may be surrounded by a reddish halo and the growth itself may show some evidence of infection or necrosis. These are sometimes called malignant papillomas.

Fig. 6. Intravenous urogram showing evidence of stasis in right kidney and filling defect in bladder caused by infiltrating carcinoma involving the neighborhood of the right ureteral orifice.

Note. Right nephrectomy was subsequently necessary in this patient.

GROUP III

In this classification are included tumors which may be pedunculated or sessile and present a definite "meaty" appearance with perceptible thickening of all or most of the papilla. They usually present evidence of infection and necrosis and may show some calcification. Thickening in the region of the base is usually indicated as a small nodular formation. This is otherwise described as papillary carcinoma.

GROUP IV

This group includes infiltrating tumors which have little or no intravesical portion, lack gross papillary formation and tend to invade the muscularis very early.

It will be noted that we do not grade these tumors on the microscopic examination of biopsy material for reasons already stated.

COURSE

The course of vesical neoplasms is generally progressive unless treated. The patient who defers treatment after the onset of symptoms greatly reduces his chances for cure. The danger increases with the frequency of recurrences, therefore the necessity of frequent cystoscopic check-ups can not be overestimated. Complications include upper urinary lesions resulting from obstruction and infection, especially in those instances where the tumor involves the region of one or both ureteric orifices. Impairment of renal function occurs in approximately one-third of the cases. Hemorrhage of a severe grade may occur. Extension beyond the bladder by infiltrating tumors to the prostate, rectum and perivesicular structures is a serious complication and inevitably tends to a poor prognosis. Very rarely, perforation into the peritoneum occurs.

TREATMENT

Since there is no way to evade this controversial subject I will express frankly my point of view. Apparently our attitude in the Middle West towards this important aspect of the problem has been too inflexible. Under the guidance of some, and more recently as a result of published reports from the Mayo Clinic, diathermy of bladder tumors has been brought to the fore, while radiation therapy has been for the most part neglected and in some clinics entirely discarded. In certain centers of the East, on the contrary, irradiation has been used almost to the exclusion of other forms of therapy. Authorities in widely separated clinics, such as Beer, Keyes, Young and Hinman, employ various types of treatment, as indicated.

Almost since its inception, surgical diathermy has been the treatment of choice in the majority of bladder cases, at the Michael Reese Hospital. Since 1928 a large number of patients treated by radium irradiation at the Edward Hines Memorial Hospital have been observed and the results of the treatment have been recorded. As a result of these experiences my impressions of this phase are as follows:

Therapy of bladder tumors affords several modes of attack, the type of the procedure depending upon the point of view and experience of the individual operator.

1. Surgical removal by resection or partial excision of the bladder wall is preferred when possible, by some authorities. This operation is neither feasible nor possible in lesions of the trigone or about the bladder neck. Those tumors which are most readily resectable, unfortunately metastasize easiest and in 75 per cent of the cases are highly malignant.

2. Total cystectomy following transplantation of the ureters into the sigmoid, is finding an increasing number of proponents. In properly selected cases it affords great comfort and prolongation of life to patients with large, highly malignant tumors.

Transplantation of the ureters without subsequent cystectomy has served excellently in a few patients with recurrent tumors of high malignancy. These procedures are indicated in some new growths of grades III and IV, especially if the involvement is extensive.

The treatment of the more usual type of tumor where the outlook for cure and comfort is happier, usually resolves itself into a choice between diathermy and radiation therapy.

DIATHERMY

The diathermic high frequency current may be used in two main ways, (1) transurethrally or through the cystoscope and (2) on the open bladder. Transurethral use of the current, is applicable to most lesions within grades I and II. Repeated cystoscopic examinations are required to confirm its effect. If found ineffective another

method should be selected. The use of the high frequency current on the open bladder is indicated in many lesions in grades II, III and IV, and in our hands has given quite satisfactory results, especially when it is the first procedure to be used. Its effect on recurrences does not seem to be nearly as great as on the primary tumor, although we have noted the exact converse of this.

By this method a certain percentage of apparent five year cures has been effected and it affords a satisfactory means of treating a large number of tumors, regardless of the degree of malignancy. I have observed a group of tumors which were resistant to diathermy and which have responded well to the interstitial placing of radium element.

It is believed by some that the beneficial results of surgical diathermy are not only due to the destruction of the neoplasm but also to its effect upon the cells of the reticulo-endothelial system, which proliferate greatly in the neighborhood of diathermized tumors. The beneficial effect of radium is interpreted in a like manner by some observers.

RADIATION THERAPY

Radiation therapy is applied either by means of the roentgen ray, or radium. There are several methods of applying radium and its emanation, two of which may be briefly mentioned; the use of the 4 gm. bomb with which we have had no experience, and the previously more popular surface application of the element. This latter method has been superseded by the more accurate and rational method of the interstitial insertion of radium or radon, which in a large number of cases has produced quite satisfactory results. To obtain the best results with this mode of treatment an adequate amount of the element or radon must be inserted well around the base of the tumor and permitted to develop its effect in accurately prescribed doses, which depends on the size and clinical grading of the tumor mass. In both hospitals the members of the Tumor Clinic cooperated and rendered technical assistance.

The insertion of radium into the opened bladder occasions practically no mortality while the patient is hospitalized. Keyes reports 37 cases treated with only 1 death. Gehrman uses radon seeds in all cases of grades III and IV tumors, while Valentine and Rogers report no cures nor apparent cures of large tumors that were highly malignant whether radium was used or not. McCarthy is even more nihilistic in his attitude toward radium, stating that he had never seen any benefits from it when it is employed in treating bladder tumors. His experience is contrary to our observations, for we have seen many new growths, including a moderately large number falling into grades III and IV, which were controlled by radium.

The value of roentgen ray therapy is probably the most controversial part of the entire discussion and my only comment is that possibly *x*-ray machines of higher voltage may be effective. Our experience does not include this type of therapy.

RECURRENCES

In the detailed records of 902 epithelial tumors published by the Carcinoma Registry of the American Urological Association, it was determined that bladder tumors recur in approximately 46 per cent of the cases. Our figures show 48.8 per cent of recurrences. Metastases occur in about 10 per cent of bladder cancers and appear oftenest in the liver. The frequency of metastases apparently does not parallel the microscopical grouping of malignancy.

RESULTS

The results of the treatment of bladder tumors have not yet reached a satisfactory level, regardless of the type of treatment used. Though the number of five year cures is increasing yearly, urologists and others are striving continually to improve this situation and encouraging signs are evident. These results have been obtained by

diathermy and radiation therapy and in a smaller group of cases by resection.

Approximately 40 per cent of the patients having papillary new growths of the bladder are alive after five years, and more than 50 per cent are alive after three years. Of those afflicted with infiltrating neoplasms, 33 per cent are alive after three years and only 10 per cent after five years.

CURES

It is difficult or impossible to determine when patients with bladder tumors are cured. Repeated cystoscopic revision is essential to their safety and well-being and these examinations should be continued indefinitely. I doubt if the expression "cure" should be used at all and I would substitute the term "apparent cure." Recurrences six, seven and eight years after operation have been recorded.

CONCLUSIONS

In conclusion I wish to stress these important points:

1. That the presence of blood in the urine must be regarded as of serious import unless otherwise determined.

2. That cystoscopic examination will reveal most of the important characteristics of bladder tumors and is essential to its appropriate diagnosis.

3. That both radiation therapy and the diathermic current afford relatively satisfactory results in many cases. Radium may control tumors which do not respond to diathermy.

4. That the results of treatment are steadily improving and many more five year survivals are reported than heretofore.

5. That an individual who has had a bladder tumor should present himself for periodic examination for an indefinite period.

6. That the future may hold increasingly promising methods of treatment which depend on none of the methods in use today, especially along the lines of biochemical and possible hormonal therapy.

I wish to thank the urological staff of Michael Reese Hospital, Dr. T. G. McDougall and members of the Tumor Clinics of both hospitals for their unfailing courtesy.

PELVIC PLICATION IN TREATMENT OF HYDRONEPHROSIS*

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NEW YORK CITY

THIS communication deals with the surgical treatment of intrinsic non-calculous obstruction at the pyeloureteric junction. An increasingly large literature and the experiments of Joelson and his co-workers[1] have had the beneficial effect of encouraging surgeons to think along conservative lines when dealing with these lesions. As a result, greater effort is now being made to preserve hydronephrotic kidneys of lessened functional capacity.

The surgical treatment of non-calculous obstructive hydronephrosis is primarily concerned with relieving the obstruction, and, secondarily, with reducing the size of the pelvis. It is not our purpose to discuss those cases of hydronephrosis in which the obstruction is due to an extrinsic cause such as an aberrant artery or external adhesions. Such kidneys, if operated on at an early stage before marked dilatation and severe parenchymal damage have taken place, often return to approximately normal function and size. However, when dealing with very large hydronephroses and those of long duration, it may be necessary to reduce the size of the redundant renal pelvis, in addition to relieving the obstruction.

On the other hand, cases of narrowing or valve formation at the pyeloureteric junction, or of high or lateral insertion of the ureter, present a more difficult problem. A variety of plastic operations has been devised to overcome such obstructions. In general, these fall into the following three classes:

1. Longitudinal incision with transverse suture of the anterior wall at the pyeloureteric junction, based on the principle of the Heinicke Mikulicz pyloroplasty (Fenger) and its modifications;[2–5]

2. Complete severance of the ureter and reimplantation into the most dependent portion of the pelvis;[6,7,11]

3. Lateral anastomosis of the ureter to the lowermost portion of the pelvis.[8]

In a recent review of this subject, Henline[9] finds that the results of the Fenger operation are, in general, unsatisfactory. This is due to mechanical buckling of the posterior ureteral wall opposite the plastic suture line. His clinical results and those of many others,[11,13,15] as well as the experiments of Herbst and Polkey,[16] would indicate that the Fenger operation, as such, should be abandoned. A modification of this operation, the "Y" plastic, has been designed to overcome these objections, and good results have been reported by Schwyzer, Foley, and others.[3–5]

The operation of severance and reimplantation of the ureter into the most dependent portion of the pelvis, that of lateral anastomosis, and their respective modifications, have been successfully accomplished in a considerable number of cases.[10,12,17,19,20,21] Lateral anastomosis, when technically possible, appears to have at least four theoretical advantages over complete severance and reimplantation.

1. The blood supply of the upper ureter is better preserved by leaving the ureter attached at its insertion into the pelvis. Its nutrition is, therefore, less impaired.

2. By the same token, interference with peristalsis of the upper ureter may be to a lesser extent in lateral anastomosis than in severance. There is a divergence

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of experimental evidence in regard to the effect of complete severance and reimplantation of the ureter on its peristalsis. Some investigators believe that section of the ureter may lead to permanent interruption of peristalsis causing urinary retention, whereas others doubt this.[22–25] Clinically, however, good emptying of the pelvis has been observed pyelographically after complete severance of the ureter and reimplantation into the renal pelvis.[10,11,20]

3. An additional line of suture is avoided by leaving the ureter attached at its insertion. This eliminates one possible source of postoperative urinary leakage.

4. A lateral anastomotic opening can be made larger, and, therefore, there is less likelihood of late stricture.

Though relief of obstruction is the primary goal in the surgical treatment of non-calculous obstructive hydronephrosis, an important secondary consideration is reduction in the size of the dilated pelvis. It is well known that such hydronephrotic kidneys, operated on early, will often return to approximately normal size if adequate drainage is provided. However, in very large hydronephroses and those of long duration, the pelvis, having lost its elasticity, frequently remains dilated and redundant, favoring, thereby, stagnation and pooling of urine. For this reason, many surgeons have attempted, in addition to relieving the obstruction, also to reduce the size of the pelvis, either by resection and suture, or more rarely by plication of its walls. Objection has been raised to these procedures on the ground that they interfere with the mechanical emptying function of the pelvis.[17] This assumption is controverted, however, by a considerable number of cases of resection described in the literature, as well as by the case of pelvic plication reported by us in this paper, which postoperatively showed normal emptying of the pelves on intravenous and retrograde pyelographic study.

The chief hazard of resection lies in the extensive suture lines which are required to repair the renal pelvis. In contrast to the intestine, the renal pelvis lacks an outer coat of peritoneum and is, therefore, less likely to heal by primary union. Any

Fig. 1. Preoperative pyelogram.

breaking down of these suture lines will result in urinary leakage and infection. Such leakage may persist in large amounts for a long time and frequently will ruin an otherwise excellent plastic repair. Nephrectomy is all too often the final procedure in such a case. In addition, it is practically often impossible to avoid perforating the mucosa when suturing a thin walled pelvis. Such sutures, when in contact with urine, may act as a nidus for calculus formation.

On the other hand, the operation of pelvic plication avoids, to a large degree, these hazards of pelvic resection. Long suture lines are eliminated, and, consequently, the danger of urinary leakage is minimized. There is also less likelihood of perforation of the mucosa by the plicating Lembert sutures.

Pelvic plication would seem to be a simpler and safer procedure than pelvic

resection. If the suture lines break down following resection, urinary leakage and infection are bound to occur, often nullifying the attempt to save the kidney. On the other hand, if the Lembert sutures in a pelvic plication do not hold, the end result, at worst, is a large dilated pelvis, for which adequate drainage has already been provided by relief of the obstruction. Obviously, pelvic plication can be of no value unless the obstruction which caused the hydronephrosis has been relieved.

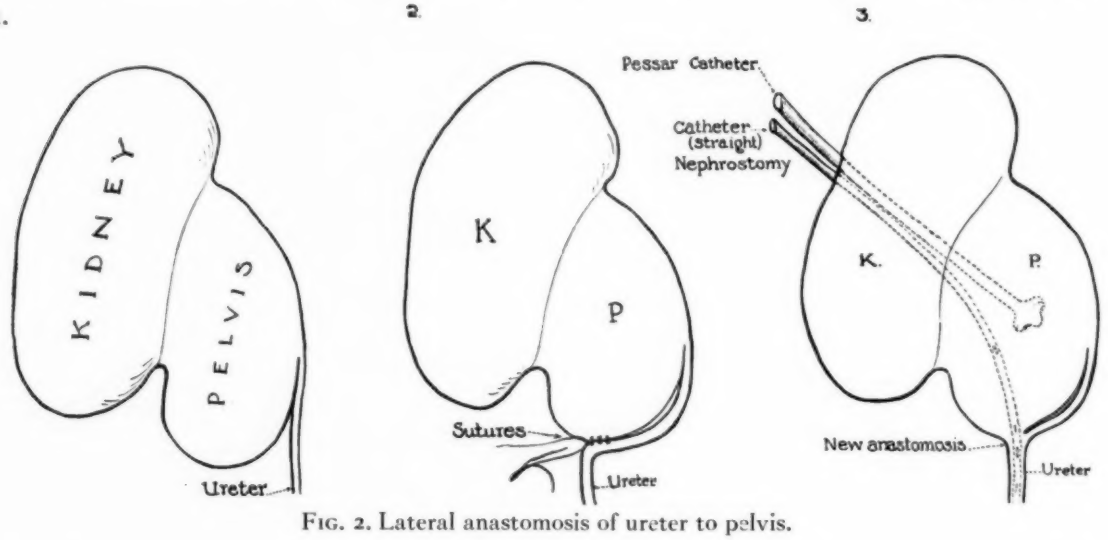

FIG. 2. Lateral anastomosis of ureter to pelvis.

Plication of the renal pelvis for the relief of hydronephrosis was first attempted by James Israel in 1894. At a later date, he[26,27] reported 3 cases in which this operation had been successfully performed. In 2 of these, an aberrant artery was the cause of obstruction, and, in the third, a so-called "S" shaped trap pelvis was found. In the first 2 cases the aberrant vessels were divided, and in the third the abnormally inserted ureter was straightened by pelvic plication and by a suture from the pelvis to the ureter. Thus its opening was relocated at the most dependent portion of the pelvis. No anastomosis was done.

A short time later, Albarran[8] reported a similar successful case, and Kelly[28] 2 others. Since then, the literature contains statements by Von Lichtenberg and Hinman[17,32] that they had each once performed pyeloplication. Mention is made of this method by others without case reports.[29–31,35] Most of these patients were operated on before the era of pyelography, and, except in Hinman's and Kelly's cases, there is no mention of any cystoscopic examination before or after operation. Symptomatically, however, the end results were reported as good.

CASE REPORT

D. W., a female, aged forty-four years, came to the Beth Israel Hospital Out-Patient Department on July 9, 1935, complaining of pain in both loins, frequent urination and pus in the urine. She was cystoscoped and a pyelogram was made (Fig. 1). Two days later the patient was admitted to the hospital because of chills, fever and severe pain in the right lumbar region. Her temperature was then 104.8°.

The next day she was cystoscoped and a moderate cystitis was found. The right ureter was catheterized 28 cm. and 100 c.c. of purulent urine was aspirated from the right pelvis. Indigocarmine, injected intravenously, appeared from the right side in twelve minutes in fairly good concentration, and in six minutes from the left. Urine from the right pelvis showed 30 to 40 white blood cells per high power field, and Bacillus pyocyaneus on culture. No tubercle bacilli were found. An indwelling catheter was left in the right pelvis. In the course of a week, the patient's temperature came down to normal. On July 18, she was again cystoscoped in order to replace

the catheter, which had become clogged. A right pyeloureterogram was then made.

Operation. On July 20, 1935, the patient's right kidney was exposed extraperitoneally through a lumbar incision. The kidney was moderately enlarged, and the parenchyma, though fairly thickened, appeared normal. The renal pelvis was greatly dilated, and the ureter had a high anterolateral insertion with definite narrowing (Fig. 2). A veil of fibrous adhesions extended over the pyeloureteric junction, causing an angulation at the point of insertion of the ureter. Three small blood vessels coursed from the pelvis to the ureter. A lateral anastomosis was made between the ureter and the most dependent portion of the pelvis over a No. 14 Fr. straight rubber catheter. This catheter was brought out through a nephrostomy opening along with a No. 24 Fr. Pezzar catheter, thus providing adequate drainage, as well as splinting of the ureter.[14] By leaving the ureter attached at its insertion, there was no interference with the blood vessels and their accompanying sympathetic nerve fibres.

The renal pelvis was then plicated after scarifying its external wall (Fig. 3). Two rows of three Lembert sutures each were passed and tied, greatly reducing the size of the pelvis. These sutures did not penetrate the mucosa. A nephropexy was then performed, fixing the kidney to the twelfth rib. The wound was sutured in the usual manner, using interrupted figure of eight chromic sutures for the muscle and fascia, and interrupted black silk sutures for the skin. The two rubber tubes, the straight catheter and the Pezzar catheter were brought out from the nephrostomy opening through the wound. In addition, a large rubber dam drain was placed near the renal pelvis.

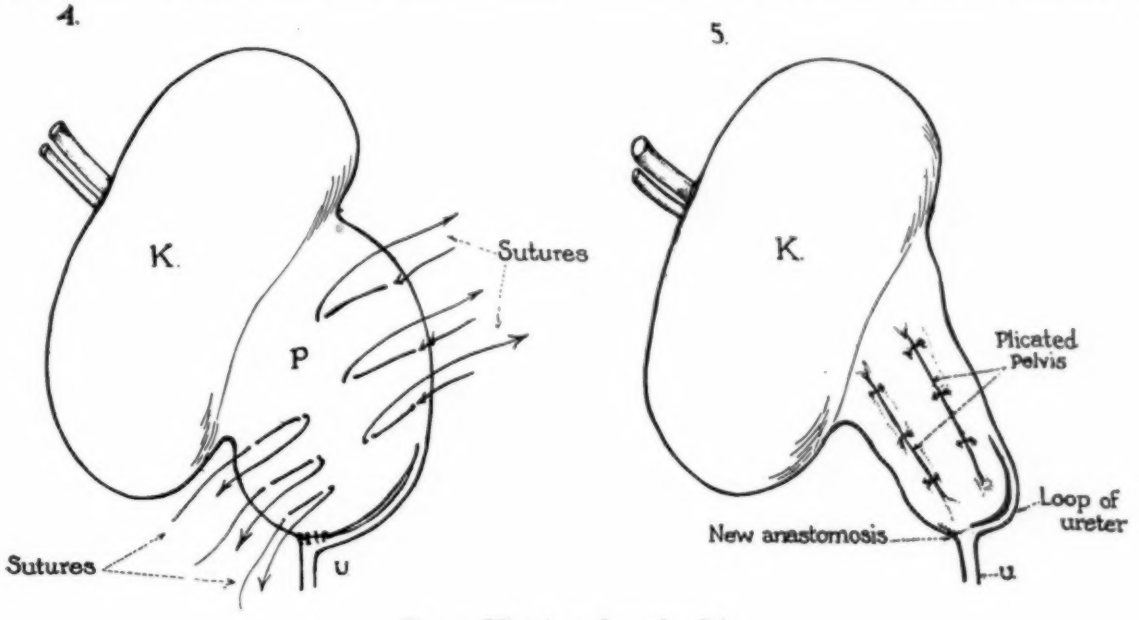

Fig. 3. Plication of renal pelvis.

Postoperative Course. The patient had an uneventful postoperative course, the Pezzar catheter being removed on the tenth postoperative day, and the straight rubber catheter on the sixteenth postoperative day.

On August 16, the patient was again examined cystoscopically. The bladder and both ureteral orifices were normal. The right ureter was catheterized 28 cm. without obstruction. There was retention of only 16 c.c. of clear urine, as compared with 100 c.c. of purulent urine before the operation. Indigocarmine injected intravenously appeared on the right side in ten minutes in fair concentration, as compared with twelve minutes in fairly good concentration preoperatively. A right pyelogram and ureterogram were made.

Two days later, the patient left the hospital with a dry wound, practically healed. She was seen in our follow-up clinic, and, on October 10 she was again cystoscoped. The bladder and both ureteral orifices were normal. A No. 6 catheter was easily passed 28 cm. up in the right ureter. There was retention of only 4 c.c. of clear urine in the right pelvis. Indigocarmine injected intravenously now appeared on the right side in three minutes in excellent con-

centration. A pyeloureterogram was made. A plate taken five minutes after withdrawal of the catheter, showed almost complete emptying of

FIG. 4. Pyeloureterogram made almost five months postoperatively; note that the ureter drains the pelvis at its most dependent portion.

the pelvis. On December 10, 1935, cystoscopy showed that the bladder was normal and there was only 2 c.c. retention of clear urine in the right pelvis. Indigocarmine appeared in three minutes from the right side, and in two and a half minutes from the left side, in excellent concentration. Microscopic examination still showed a few red blood cells and white blood cells in the urine from the right pelvis. Bacillus pyocyaneus was cultured from both sides. A right pyeloureterogram (Fig. 4) and retention plate were made. There was complete emptying of the injected sodium iodide solution ten minutes after the catheter was withdrawn.

The patient was last seen on April 9, 1936. She had no urinary complaints and felt well. An intravenous pyelogram showed excellent function of the right kidney.

COMMENT

This case demonstrates a good functional and anatomical result following lateral anastomosis of the ureter to the most dependent portion of the renal pelvis, combined with plication of the pelvis after external scarification of its wall. The pelvis was restored to approximately normal size and emptied freely. This occurred despite the presence of infection at the time of operation. Obviously, the calyceal dilatation, which was present before operation, was little, if at all, influenced. However, the function of the affected kidney, as evaluated by intravenous indigocarmine had greatly improved. One could reasonably predict that the operated kidney could sustain life in the event that it were necessary to remove the other kidney.

Having had the good fortune to obtain this clinical result, we attempted to carry out animal experiments in order to determine if firm healing would regularly take place, following scarification and plication of a dilated pelvis.

EXPERIMENTS

A dozen rabbits were used but, due to infection, shock and intercurrent disease, only 3 of these survived the entire experiment.

At a first operation, the right ureter was exposed intraperitoneally at its midportion, and ligated in order to produce hydronephrosis.[33,34]

On the seventh, twelfth and twenty-first days, respectively, the right kidney was exposed transperitoneally. Extraperitoneal exposure was attempted several times, but was found impossible due to the thinness and friability of the peritoneum. One or two fine silk Lembert sutures were placed in the dilated pelvis, plicating it. A lumbar nephrostomy was made. In our early experiments we attempted to relieve the ureteral obstruction by removing the ligature, but found this technically impractical. The ureter was easily torn, since considerable exudate and adhesions were present at the site of ligature.

The nephrostomy tubes were left in situ from seven to ten days until a competent drainage tract had formed. The animals

were autopsied ten, sixteen and seven days, respectively, following the second operation.

The site of plication in rabbits 1 and 2 was examined histologically by Dr. Alfred Plaut, pathologist, who reported, as follows: "Cross sections, through the areas in the kidney pelves indicated by the surgeon, show ligatures in situ. In the region of these ligatures, the tissue is thick and fibrous. There is no inflammation."

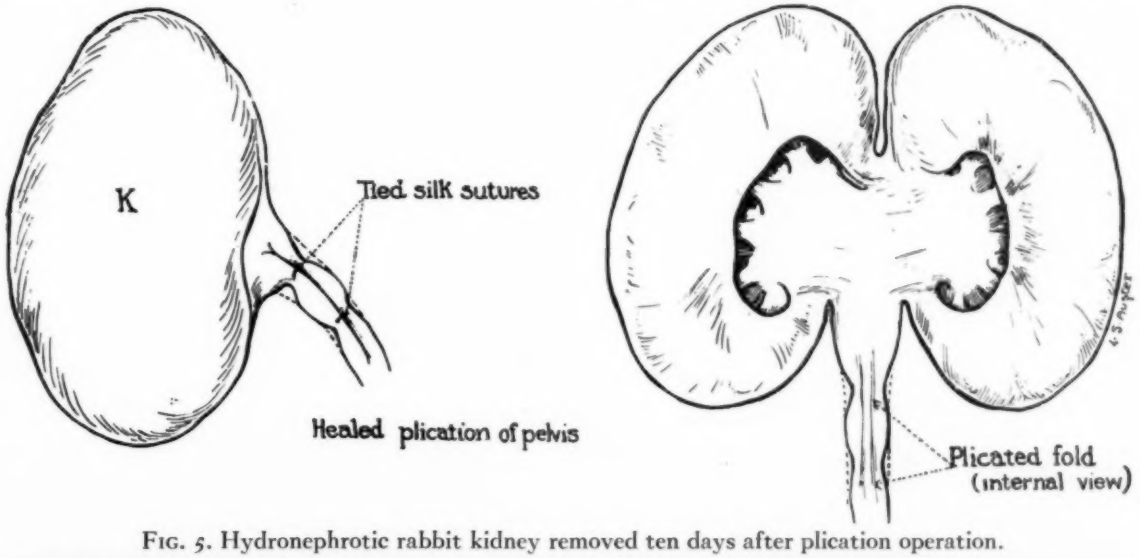

Fig. 5. Hydronephrotic rabbit kidney removed ten days after plication operation.

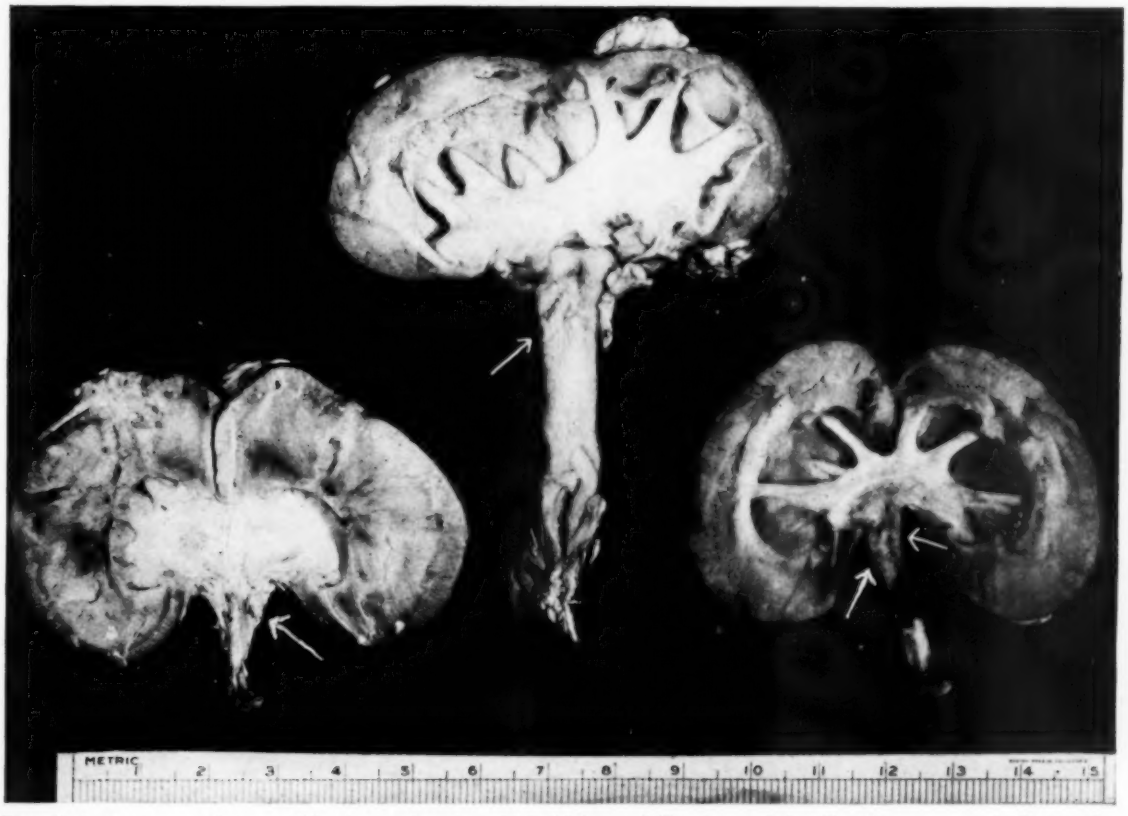

Fig. 6. Autopsy specimens of hydronephrotic rabbit kidneys following pelvic plication; arrows indicate the healed ridges formed by plication

The pelves of all three animals showed a distinct firmly healed ridge at the site of plication (Figs. 5 and 6). Healing had occurred, despite the evidence of considerable gross infection at autopsy in every case.

From these specimens, it would appear that plication of a hydronephrotic pelvis, following scarification of its wall, results in firm fibrous healing in the rabbit.

CONCLUSIONS

1. The surgical treatment of non-calculous obstructive hydronephrosis has two aims.

a. Relief of the obstruction, thereby providing adequate urinary drainage; and

b. Restoration of the dilated pelvis to relatively normal size and contour.

2. Relief of obstruction in cases of intrinsic stricture at the pyeloureteric junction, or of abnormal insertion of the ureter into the renal pelvis, may be accomplished by a variety of plastic operations. Lateral anastomosis of the ureter to the most dependent portion of the renal pelvis, when feasible, is probably the operation of choice.

3. Plication of the renal pelvis, following scarification of its external wall is a simple and safe method for reducing a markedly dilated pelvis to approximately normal size and contour. There is less likelihood of postoperative urinary leakage, or of late calculous formation, as compared to resection and suture of the renal pelvis.

4. Pelvic plication is of little or no value unless the obstruction which caused the hydronephrosis has been relieved.

5. An operated case and the results of animal experiments are reported as evidence that plication of the pelvis will heal firmly, despite the presence of urinary infection.

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DISCUSSION

DR. OSWALD SWINNEY LOWSLEY. This is an excellent method, and the author has brought out the various essential points, and empha-

sized very properly the fact that the most important part of the operation was to relieve the cause of the obstruction. Sometimes that is relieved by dilatation, but usually when the case gets thus far advanced it is relieved by operation. Very frequently obstructions may be relieved by dilatation, and no one knows that better than I do from personal experience. I feel that Dr. Wilhelm was perfectly justified in operating on this case as early as he did. I think in many of the cases in which plications were done for hydronephrosis we have waited too long, and that is borne out by the fact that so many of the plications which we have done and others have done have had to have nephrectomy following the plication. This happens all too often, the reason being that a stricture forms at the site of the operation, regardless of the method. We have every reason for a stricture to form there. We have infected urine, because these cases are practically always infected, and nothing lays down scar tissue more actively than infected urine, and all too often these plications are followed by nephrectomy. As a matter of fact, this is such a truism that we have come to the point of doing a pyelotomy for stone very rarely, unless there is a very large pelvis which makes us pretty sure that a stricture will not result, with loss of the kidney. This is particularly true since our methods of performing nephrostomy have improved.

DR. STANLEY R. WOODRUFF. For a number of years, I have been trying to bring out that there is no such disease as hydronephrosis; it is a condition. Pyonephrosis is a disease, but hydronephrosis is not a disease and we should speak of it as obstructive pyelectasis. I believe a large part of operative, plastic procedures on the renal pelvis are probably unnecessary, because as a rule, in fact practically always, they are due to some obstructive condition, and Dr. Wilhelm himself admits the first thing to do is to remove the obstructive condition. In nearly every patient who suffers from such a condition caused by obstruction the kidneys are ptosed, and in my experience, if the obstruction is removed and the kidney is fixed in a proper high position, I believe that practically all of them will empty successfully. It has been my experience that they have. I do not remember ever having plicated a pelvis or done any plastic work on it, although once in removing a large stone from the kidney when I got through I found I had also removed the ureter and I sewed the ureter back onto the renal pelvis. I had the luck of my early days of surgery, which I probably would not have now, that there was no bad result from it at all. The ureter healed perfectly into the kidney pelvis. I believe if you remove the obstruction and put the kidney into the proper position where it is going to drain that such a procedure will be sufficient.

DR. JOHN H. MORRISSEY. I did not have an opportunity of reading Dr. Wilhelm's paper before coming to this meeting, and as a result have prepared a short discussion embodying my feelings about the general subject of reshaping a kidney pelvis, without a knowledge of his findings and conclusions. My own experience has been limited to some 7 or 8 cases and has not been too entirely satisfactory inasmuch as 3 came to subsequent nephrectomy. However, I am frankly amazed at the precocity of Dr. Wilhelm in endeavoring to draw clinical conclusions from one hospital case operated on and a couple of rabbits. As you may know, the rabbit's pelvis measures something like 2 by 0 and how any extensive experimental procedures could be carried on from which value bearing data might be assembled is more than I know. After all, the success of an operation of this sort depends upon the disappearance of subjective symptoms of hydronephrosis, whatever they may be. It would seem that the present study assumes anomalous vessels to be the main factor in hydronephrosis, while the studies of Mr. Winsbury-White on this subject point to the contrary. As a matter of fact, he seems to think infection in the lower tract plays a major role in the gradual dilatation of the pelvis, both in the male and female, and under such conditions a plication procedure would be by no means indicated.

Then too, how is one to gauge the clinical success of this method? Does it depend upon the disappearance of symptoms, whatever they may be, or as one might draw from one enthusiastic investigator in this field, does it depend upon the ability of the kidney to tolerate forty finely placed sutures at micrometric distance from each other? Frankly I am hostile to these fine techniques, savoring of a lacemaker, and feel rather that when sufficient attention and study have been given to the element of focal infection,—low placed kidney—and infection within the kidney pelvis and parenchyma in these cases and results are not obtained and symptoms persist, then nephrectomy is indicated, especially in the presence of a lessening function, and of course, a good second kidney. I have not found essential

undue and extreme niceties of technique in reshaping of the pelvis; interrupted or running catgut sutures through the pelvis are sufficient, followed by retrograde dilatation of the ureter and most of all, fixation of the kidney at an adequate level. These are to my mind of just as much importance as the plication.

DR. THOMAS J. KIRWIN. Some years ago I spent a considerable time working on the problem of ureteral transplantation and thought I had hit upon something quite original and previously unheard of, that was that the ureter, once traumatized, never completely recovers its tonus, and as a consequence ureteral peristalsis remains impaired and in most instances, is permanently inhibited. My claims to originality were short lived, however, for a little research showed that as far back as 1908 Alkane, a German, had discovered the same thing, and twelve years later, Marion's assistant, Gouverneur, in France, had extended the research and showed by animal experimentation and the clinical records of patients whose ureters had been repaired after traumatic accidents, that peristalsis could not be restored 100 per cent.

Thus chastened, I turned my attention to investigating the nerve supply of the ureteral wall. I succeeded in proving that there really are ganglion cells there which give off fibers running into the muscular tissue. Satani showed that the ureter's outer coat contains a wrapping of unbroken nerve networks, including ganglia on both sides. The fibers running from them make connection between the two plexuses in the outer fibrous and inner mucous coats. They are thus conductors of nervous impulses to the muscle tissue.

If any of these outer "nerve networks" are cut they can never fully recover their tone, no matter how perfectly healing of the severed ureteral tissue may seem to have occurred. I admit that some of the results obtained by Beer and some others would seem to contradict this. But I still feel sure that sooner or later peristalsis will be impaired, and hydronephrosis with eventual destruction of the kidney will be the end result. All the plastics done on the renal pelvis will not get around this, and in the end the kidney will have to come out, as Dr. Morrissey has said.

DR. SEYMOUR F. WILHELM. I want to say only a few words, and one of them is that Dr. Lowsley brought out a very important point when he said that, if these cases are operated on, they should be operated on early, before severe parenchymal damage has taken place. If the kidney is already greatly compromised, there is little chance of any considerable return of function after plastic operation.

I appreciate Dr. Morrissey's and Dr. Woodruff's remarks very much, because I am in agreement with them to a large extent. It is true that a great deal too much plastic work may be done on the pelvis. I tried to emphasize and make clear that the important thing in the treatment of obstructive hydronephrosis is to relieve the obstruction. We were dealing only with cases of intrinsic obstruction, hydronephrosis due to true stricture at the pyeloureteric junction. As I said before, I am not discussing the treatment of cases of atonic hydronephrosis nor of obstructive hydronephrosis due to external causes.

In this particular group of cases, that is, hydronephrosis due to intrinsic obstruction at the pyeloureteric junction, the pelvis will often return to approximately normal size and contour if the obstruction is relieved. It is quite unnecessary and improper to do any type of pelvic resection or plication on such pelves. There are, however, certain cases in which the pelvis is very large and has lost its elasticity. In such cases there is a good deal of redundancy, which tends toward urinary stagnation, and as a result, late calculus formation. In this type, only, is it justifiable to attempt any type of plastic repair, such as plication.

One point made by Dr. Morrissey is particularly good; that is, if too many sutures are placed in the renal pelvis it will lead to trouble later. It is for just such reason that we have adopted pelvic plication, a simpler and a much safer procedure than resection, which requires such extensive suturing.

The question raised, as to whether plastic operations should ever be done, or whether nephrectomy should always be performed on these large hydronephroses, we cannot decide tonight. Some of us have the feeling that many damaged kidneys may be saved and restored to good function by conservative operations. I do not propose to lay down dogmas on the basis of a single case, or of a few animal experiments, but I do feel that by showing single cases like this, with thorough cystoscopic and pyelographic follow-up, we can accumulate a large experience. It is from such a collection of reports that we can really find out whether plastic operations on the pelvis, based on sound surgical principles, will preserve kidneys for any appreciable length of time.

DANGERS OF INTRASPINAL (SUBARACHNOID) INJECTION OF ALCOHOL: THEIR AVOIDANCE AND CONTRAINDICATIONS*

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NEW YORK CITY

THAT the injection of 95 per cent or absolute alcohol into the spinal subarachnoid space for the relief of intractable pains and for sympathetic nervous system disorders is accompanied by the risk of bladder and rectal complications, and even motor paralysis or death has been stated repeatedly in my previous articles.[1–7] Dogliotti and others[8–13] have also warued of possible complications. Aird and Nafziger[14] as well as myself[4] have shown some of the pathological changes which may follow the experimental intraspinal injection of alcohol in cats. We have stressed the narrow margin of safety in dosage and technique in using this method of relieving pain.

While the insertion of a spinal puncture needle into the lumbar subarachnoid space should be a relatively simple procedure, the introduction of a needle by the inexperienced may be accompanied by trauma to the nerves of the cauda equina. Following such injury the effect of any introduced alcohol is enhanced many times. This was clearly shown when desoxycholic acid was combined with alcohol in minute quantities and injected into cats.[15] Relatively large amounts produced no motor complications while very small amounts caused paralysis and severe sensory disturbances when the nerve tissue was first traumatized. This was brought to my attention when an attending surgeon asked me to "stand by" while he performed his first intraspinal injection of alcohol to relieve a patient of pain arising from a prostatic carcinoma. He proceeded to insert the needle in the midline of the back between the third and fourth lumbar spines but for some reason or other no spinal fluid could be obtained, although the needle was poked into the spine about twelve times without success. Following this, without any difficulty at all, I obtained slightly blood tinged spinal fluid on my first attempt. I did not realize at the time that the injection of alcohol following trauma such as unquestionably occurred in this case, would be followed by complications. I had done dozens of similar injections before and had had no complications. However, following the injection of 10 minims of absolute alcohol this patient developed bladder retention and rectal incontinence. Undoubtedly, the traumatized nerves allowed the small amount of alcohol to infiltrate and cause more damage than was anticipated.

While the introduction of a spinal needle is relatively simple in the lumbar region, it is somewhat more difficult to insert in the thoracic region, especially the upper section, on account of the length and inclination of the spinous processes. Moreover, the size of the interspaces is normally much smaller than in the lumbar region and in adults with mild or moderate degrees of productive osseous changes of their vertebra, these interspaces may be considerably reduced or even obliterated completely by the bony fusion of the dorsal plates.

The injection of the prescribed dosage of alcohol following the accidental puncture of the spinal cord, or injury of the spinal blood vessels may cause complications. It is therefore advisable to refrain from injecting any alcohol whenever there is difficulty in getting clear spinal fluid, or where a slightly bloody fluid is obtained which does not clear after the removal of a

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few cubic centimeters of fluid. Unless the needle point is unquestionably in the subarachnoid space and there is a free flow of fluid, or, in cases of low pressure where fluid can be obtained by having the patient cough, or on compressing the jugular veins, or upon aspiration with a small syringe, alcohol should not be injected under any circumstances.

As a result of a previous injection, or due to some other process, one may have difficulty in obtaining fluid because of an adhesive pachymeningitis causing a complete or partial blockage. While the first injection may have been done with perfect ease, subsequent attempts at this same level may prove futile. One may then get a free flow either one space above or below this level without difficulty. It is readily conceivable that in such a case, puncture of the cord may be done inadvertantly, if the possibility of such a condition is not kept in mind. Symptoms of a partial or complete transverse myelitis may follow, even without the injection of any alcohol.

Occasionally in the upper thoracic region the spinal fluid is held in relatively closed meshes by the arachnoid membrane, especially in the region of the dorsal suspensory ligament of the cord which is considered a reduplication of the arachnoid membrane. It is conceivable that the needle point may enter such a locus and fluid be obtained. Upon injection, the alcohol may become enmeshed in this ligament and remain in a concentrated form instead of becoming diluted as occurs when injected in the "free" subarachnoid space. The approximate active percentage of alcohol as it comes in contact with the nerve roots is estimated to be 20 to 25 per cent when two minutes is consumed in injecting 16 minims of absolute alcohol without barbitage. If there is interference with the normal diffusion rate, one can readily understand how a more concentrated solution of alcohol will produce more marked affects than anticipated.

Repeatedly, I have drawn attention to the fact that more than 8 minims of absolute alcohol injected between the second and third lumbar spines will invariably cause bladder complications, whereas above or below this level, as much as 16 minims have been injected without bladder or rectal complications ensuing. This was vividly brought to my attention when I tabulated and summarized the results of my first fifty injections.[2]

Others found the same complications. Upon further investigation, almost invariably, more than 8 minims was found to have been injected between the second and third lumbar spines. This explains the complications reported by Tureen and Gitt[13] in their Case 11.

The bladder complication which may occur results from a sensory rather than a motor paralysis, as undoubtedly at this level the sensory nerves from the bladder are affected, the bladder becomes distended and dribbling occurs from the overflow. In some cases, this distention and retention may not be recognized by patient or physician, and back pressure and infection may follow. As seen in experimental animals, a pyonephrosis may develop, with marked dilatation and ulceration of bladder and ureters, which may be fatal in time. Bladder retention with injection at this level may occur despite the fact that only one side of the vesical innervation is affected. Occasionally bladder retention may continue only twelve to forty-eight hours. Sterile cathererization and the administration of urinary antiseptics are indicated when retention persists.

With the larger doses of 16 minims of alcohol injected between the third and fourth, or fourth and fifth lumbar spines, rectal incontinence may follow, especially when the hips are elevated too high. This allows for the alcohol to "fill" the entire lower end of the dural sac and as a result the lower sacral and coccygeal nerves of both sides are affected. Complete anal anesthesia follows and the sphincters usually become relaxed so that the patient loses all sensation and soils the bedclothes.[16] In other cases the sphincters retain their

tone and only sensation is lost, so that the rectum and colon become distended with feces. Such a condition if unrecognized, may cause a marked toxemia from auto-intoxication, intestinal obstruction and death. Such retention warrants the use of daily enemata.

I have observed repeatedly that chronic constipation has frequently disappeared completely in patients receiving intraspinal alcohol injections for the relief of intractable pain. I believe that alcohol at the proper levels may affect the sympathetic nerves of the colon and rectum so as to remove the inhibiting affect of these nerves. What is accomplished by sympathectomy for Hirschprung's disease, may therefore well occur with the intraspinal injection of alcohol. Naturally, only those cases of "atonic" constipation would be benefited in this way.

With only unilateral desensitization of the anus, rectal incontinence does not occur. It is therefore important not to elevate the buttocks of the patient too high. A maximum dose of 10 to 12 minims of absolute alcohol should be injected in the last two spaces. This may explain the complication in Case 1 reported by Tureen and Gitt.[13]

Since the introduction of this method of relieving pain, a number of variations in technique have been used. While good results may be obtained by keeping the patient in position only fifteen minutes after the injection has been completed, some have sought to accentuate the effects by keeping the patient in position for three to six hours[16] and even twelve hours. This is apt to allow the alcohol to remain "undiluted" too long and complications may result. Patients should not be kept in the lateral position for more than one-half to one hour at the most. This probably helps explain the complications in Case 1 by the mentioned authors.

There is a finesse in securing the proper degree of action which can only be obtained by experience. Too great a concentration, too large a dose, or the maintenance of the lateral position too long may cause more marked effects than desired. Too small a dose, insufficient concentration, or more rapid diffusion may cause no effects at all. In either event, a single experience of failure to relieve pain, or an unexpected bladder or rectal complication should not cause any reflection on the efficacy of the method. There is no question of the value of this method to relieve intractable pain, as evidenced by numerous reports appearing in increasing numbers.[7] Personal experiences with several hundred of these injections convinces me of its value.

Occasionally the doctor is confronted with the patient suffering agonizing pain and who with the family, expects miraculous relief from a single injection. The doctor may be inclined to inject an extra good dose, only to be chagrined by the occurrence of unpleasant complications. It is much better to explain to the patient and his family that occasionally more than one injection may be necessary, especially where pain is bilateral or over more than three segments. It is also advisable to inform them that relief may not come until the lapse of several days, or even one, two or even three weeks. In any event it is much better to repeat the injection with small doses, to "feel one's way," and be guided by the sensory affects of each injection than to attempt to cover too wide an area with a single dose. When relief does come immediately and completely, the effect is miraculous and when it lasts for twelve to eighteen months, the patient is most grateful. The need for more than a single injection in some cases has not been emphasized in previous reports.

Another deviation that has been tried by others with resulting bladder or rectal complications is to turn the patient on his abdomen instead of his back after the injection is completed. This tends to affect both sides, so that too marked an action is obtained on the afferent pathways.

When motor weakness or "paralysis" follows an intraspinal injection, providing no damage is done to nerve tissue by the needle, these complications are usually

transitory and depend again on the dosage and concentration. This motor paralysis is not due to a direct action on the anterior roots when the smaller doses are used. Motor weakness or paralysis may follow only the interruption of muscle, tendon and joint sensations, without the interruption of the anterior or motor roots.[16] Just as with ordinary spinal anesthesia, the patient is unable to move his limbs because the anesthetic solution has affected the dorsal roots only,[17] so with the larger doses of alcohol, all afferent sensations may be interrupted, causing a more permanent anesthesia with weakness or disability. This condition usually clears rapidly unless so much alcohol has been introduced that the anterior roots or spinal cord itself have been affected. This may result in a true motor paralysis, accompanied by trophic ulcerations, bladder and rectal complications, inanition and death.

Occasionally a chemical meningismus follows an intraspinal injection. This is the exception. The patient develops pains up and down the spine, headache and slight rigidity and retraction of the head which may become alarming. The spinal fluid cell may reach 20 to 30 per cu. mm. in twenty-four hours, and the fluid itself be under pressure. Treatment by repeated puncture may be indicated, as well as sedatives and cold compresses to the head. This condition may last for one to four days and may set in only after three days have elapsed following the injection.

Alcohol taken at random may contain spores. Only sterile alcohol filtered and boiled under pressure should be used. This can be obtained in ampoules.

While respiratory paralysis and death may occur theoretically in humans if allowed to sit up immediately after spinal alcohol injection, no such complication has been called to my attention. Experimentally, if one allows even a few minims of alcohol to come near the medullary centers, respiration may cease and after fifteen to twenty minutes the heart stops beating. It is therefore imperative for the doctor to be responsible personally for the maintenance of the patient's position with the head kept low. While patients have gotten out of bed two hours after the injection, this procedure is not advocated since severe headaches are apt to follow. It is therefore better to see that the patient is thoroughly evacuated and the bladder emptied before the injection is started. After three hours have elapsed, the patient may safely sit up but should be kept quiet in bed for at least twenty-four hours. Following the three hours, in exceptional cases, lavoratory privileges may be allowed, but the use of the bedpan and urinal is preferable.

Following an injection of alcohol, the patient sometimes perspires profusely and therefore should be protected from draughts and well covered.

As a rule, the procedure is unaccompanied by any pain or shock. Should the patient feel weak, stimulants may be given by a drinking tube without elevating the patient's head.

In one case of intractable asthma, the patient developed a typical shock condition about six hours after the injection.[6] The patient had been under the constant influence of adrenalin before the injection. Following the alcohol injection, her asthma promptly stopped, and the adrenalin was discontinued. The sudden withdrawal of adrenalin may have induced the shock reaction. The patient, however, promptly responded to stimulants of adrenalin, caffeine and intravenous glucose solution.

In determining the dosage, an important point to be kept in mind is the fact that the adult female spinal canal may be smaller than the adult male spine. One should use somewhat smaller doses in the female in order not to get too profound an effect.

CONTRAINDICATIONS

Patients with very extensive ulcerating cancers are apt to bleed after intraspinal alcohol injection. The fear of this, however,

should not prevent giving the sufferer relief in most such cases.

Patients with diabetic or arteriosclerotic gangrene of the extremity may get relief from this procedure, but if infection is present it may spread more rapidly after alcohol injection, due to the increased vascularity. Ordinary necrosis due to gangrene, without spreading cellulitis, may demarcate and heal completely after intraspinal injection of alcohol, even when bone is exposed and infected.

Patients with polycythemia may develop an increase in their red cell count following injection. In a patient who is apt to have embolic phenomena, this must be borne in mind. I have found that the red cell count may increase as much as two million in twenty-four hours and the hemoglobin increase twenty per cent. The exact cause for this has not been determined. An interesting finding has been an increase in the eosinophiles in some cases to as much as twelve per cent in twenty-four hours, while the total white cell count remains unchanged. The exact significance of this also is not known.

Certain allergic cases may react badly when alcohol is injected intraspinally. While the amount of protein normally in the spinal fluid is small, the alcohol may precipitate some and change its chemistry so as to cause a reaction. The possibility of the Schwartzmann reaction occurring intraspinally should also be investigated. The case of asthma cited had a profound sensory anesthesia with transitory bladder and rectal incontinence and difficulty in moving the lower limbs. This might have been caused by an allergic reaction since the technique of the injection was perfect.

Injections should not be made through infected areas or metastatic deposits.

Acute surgical conditions or lesions requiring surgery should not be injected, unless the condition is hopeless and operation impossible.

In any obscure case, injection should not be given until all methods of investigation have been tried. Spinal cord tumors should be especially kept in mind. The removal of pain as a symptom may make the diagnosis more difficult on the one hand, but may shift the pain closer to the site of the lesion on the other. Referred pains may travel via the sympathetic nerves, which when blocked at one level, may enter the cord at a different level. This was most interestingly demonstrated in a patient with upper left sided chest pains. When blocked, the pain centered over the left kidney and *x*-ray examination then revealed a small stone in her right kidney which never had caused any urinary signs or symptoms.

The injection of alcohol should not be done above the first thoracic vertebra on account of the danger to the phrenic and medullary nerve centers.

One should not expect this method to relieve every case of cancer pain; no procedure is 100 per cent perfect. Pains arising from lesions in bones are more difficult to relieve than pains arising from soft tissue cancers. Repeated injections often stop pain where the first injection has had no effect. One should not be ready to condemn this method as to its efficacy from a single failure.

Drug addicts may continue to complain of pain in spite of alcohol injections. Chronic sufferers should therefore be injected before they become drug addicts.

SUMMARY

1. Bladder and rectal incontinence are the most frequent complications after the intraspinal injection of alcohol but can be avoided by following accurately the details of technique and dosage. Modifications of technique are accompanied by danger. No more than 8 minims should be injected between the second and third lumbar spines.

2. Bladder and rectal complications can be avoided by limiting the action of the alcohol to one side at a time.

3. Loss of muscle, tendon and joint sense may cause somatic motor "paralysis," which is usually transitory.

4. Excessively large doses may cause true motor paralysis by affecting the anterior roots or even the cord itself.

5. Trauma to nerve tissue at the time of the injection increases the action of the alcohol.

6. Patients should be kept with the head lowered, else medullary complications may occur.

7. Repeated smaller doses should be used rather than a single large dose.

8. Infections of the spinal column not well localized, extensive bleeding cancers, diabetic or arteriosclerotic gangrene accompanied by spreading cellulitis, polycythemia vera, acute surgical conditions or chronic lesions requiring surgery should not be injected. Where surgery is contraindicated or the case is hopeless the pain may be relieved by this method.

9. Every attempt should be made to find the cause of obscure pain before resorting to this method of relief. Following the relief of pain every effort should be continued to arrive at a positive diagnosis.

10. Drug addicts may not be helped by spinal injections. Chronic sufferers should receive their injections before they become habituated to narcotics.

11. Intraspinal alcohol injections should be given only by those possessing a knowledge of the physics of the method, and the details of the anatomy, especially of the sympathetic nervous system.

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CASE REPORTS

CARDIOSPASM

REPORT OF TWO CASES TREATED BY RESECTION OF SYMPATHETIC SUPPLY TO THE CARDIAC SPHINCTER*

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THIS condition was first described by Purton in 1821 as idiopathic dilatation of the esophagus. Since then various names have been applied to it in an effort to have a descriptive, etiological term. Mikulicz popularized the term cardiospasm, postulating the theory that the obstruction was due to spasm of the cardiac sphincter at the lower end of the esophagus. This has become the generally used term in the literature for the "condition (quoting Walton[45]) of dilatation and hypertrophy of the esophagus, where on postmortem examination no obstruction to the lumen can be found distally." Phrenospasm (Jackson[16]), achalasia (Hurst[15]), esophagectasia (Batty, Shaw and Woo[2]) and preventriculosis (Jackson) are synonymous terms.

PATHOLOGY AND ETIOLOGY

The most characteristic pathological change is the huge dilatation of the esophagus. Moore,[29] Shattock,[40] Brown[3] and Rake[34] have studied the gross and microscopic pathology. The esophagus may assume one of three characteristic forms, fusiform, flask shaped and sigmoid shaped. These are probably degrees of the same process, the sigmoid shaped dilatation being the terminal stage, where the esophagus not only is dilated but has lengthened. Always accompanying this huge dilatation there is a commensurate degree of hypertrophy of the muscle wall. Occasionally the cardiac sphincter appears to present distinct hypertrophy. Such instances are exceedingly rare. Because of the fact that definite esophageal obstruction can be demonstrated pathologically they probably should not be classified as cases of cardiospasm in the manner that Walton conceived of this term.

In advanced cases, secondary to stagnation of food, an ulceration and quite often an inflammation of the lower end of the esophagus is seen. MacCready[24] reports small white patches of epithelial proliferation or leucoplakia in one specimen. Rake[35] also found definite evidence of malignancy in 3 of 15 fatal cases.

Many factors have been advanced as etiological agents. Mosher[30] suggested some deformity of the liver tunnel. However, diseases producing enlargement of the liver and one might assume therefore more likely to encroach on the liver tunnel, do not result in esophageal obstruction. Batty, Shaw and Woo[2] suggested that the dilatation was due to kinking as a result of the extreme length of the esophagus. Most people agree that the increase in length of the esophagus is a result of cardiospasm rather than a cause. Jackson[16] offered the theory that the obstruction was due to improper functioning of the "diaphragmatic pinch-cock" and suggested the term phrenospasm. However, though most of these obstructions do occur where the esophagus passes through the diaphragm, Watts[46] reports a case where operation revealed dilatation in the esophagus below the diaphragm.

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The present concept as expressed by Einhorn,[5] Rolleston[39] and Hurst[15] offers the hypothesis of failure of adequate relaxation at the cardia. On the contrary, without difficulty while others allege that one feels a definite spasm of the cardia, is confirmation that clinical as well as physiological opinion is still disunited.

FIG. 1. Case 1. Film taken January 9, 1935, showing complete obstruction at the cardia. Esophagus does not fill evenly with barium because of the large residue of food.

FIG. 2. Film taken May 5, 1936, still showing marked esophageal dilatation. However, a thin stream of barium can be seen entering the stomach. Several peristaltic waves are also seen.

Walton[45] still feels that an abnormal degree of spasm is the most adequate explanation of the observed pathological physiology. The fact that the endoscopists divide themselves, some maintaining that bougies can be passed by the site of obstruction

In support of the theory of achalasia, or failure of relaxation, Rake[34] cites the following histological observations. He was able to show definite inflammatory changes involving Auerbach's plexus. The ganglia

are increased in size and show marked cell infiltration, lymphocytes being in predominance, together with a few eosinophiles and mast cells. In the later stages one sees a disappearance of the ganglion cells and commencing fibrosis of the plexus. Rake was able to demonstrate this in every one of 11 cases. Cameron found the same microscopic picture in 8 cases and Mosher and McGregor in one. Freeman,[9] however, was not able to demonstrate this involvement of Auerbach's plexus in his case.

Kronicher and Meltzer were able to produce a condition of achalasia with secondary esophageal hypertrophy and dilatation in rabbits by division of the vagus nerves. Jordan[17] reports that a section of the vagus causes increased symptoms. Rieder[37] also found that the dysphagia in a patient was aggravated by section of both vagi. Poulton and Payne[32] were able to demonstrate relaxation of the cardiac sphincter in the rabbit by stimulation of the vagus. The experimental work of Knight[19] on cats, so clarified the pathological physiology that one is justified in abstracting his conclusions.

By stimulation experiments it was shown that there is a true intrinsic sphincter at the cardiac which is relaxed by the vagus and contracts on sympathetic stimulation. The sphincter is capable of functioning independently of surrounding structures and receives its sympathetic supply from the celiac plexus in fibres which follow the course of the left gastric artery and its esophageal branch to the lower end of the esophagus.

Excision of the extrinsic nerves supplying the esophagus as studied by skiagrams taken of bismuth meals, showed that vagal excision reproduced the *x*-ray, pathological and clinical picture of achalasia of the cardia, the sphincter failing to relax in the course of peristalsis, if, however, both thoracic sympathetic chains were excised at the same time as the vagi were divided, no obstruction resulted at the cardia. The activity of the sympathetic is therefore essential for the obstruction to occur. Denervation by the operation of celiac sympathectomy of the sympathetic fibres described above as supplying the cardiac sphincters resulted in a complete loss of sphincter tonus so that the meal could be caused to regurgitate from the stomach to esophagus. Later the tonus recovered somewhat but there was abnormally rapid passage of the meal through from esophagus to stomach.

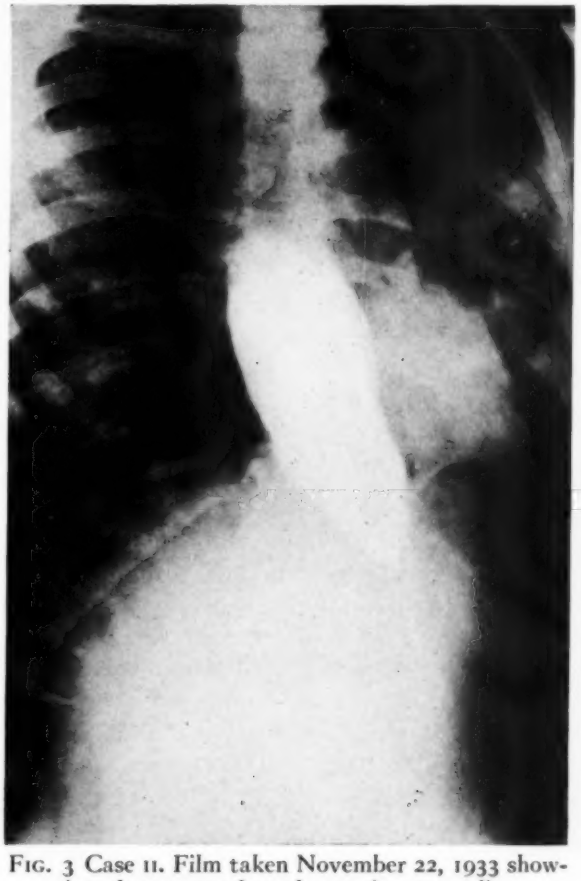

Fig. 3 Case 11. Film taken November 22, 1933 showing almost complete obstruction at cardia.

In animals in which achalasia of the cardia had been produced by vagal section, subsequent celiac sympathectomy resulted in a complete relief of symptoms and skiagrams showed the sphincter relaxing and the meal entering the stomach without obstruction. The success of sympathectomy is to be judged on this relaxation of the sphincter rather than on regurgitation from stomach to esophagus which disappears as the muscle recovers tonus after the operation.

A criticism of these results is that the vagal lesion caused experimentally did not affect the intrinsic neuromuscular mechanism of the esophagus. In man it may be that lesions of Auerbach's plexus as described by Rake might interfere with the recovery of the tonus, and peristalsis in the esophagus. If this be so, one

would expect greater benefit to be derived from the operation in cases of a spasmodic type with a normal esophagus than in cases in which

FIG. 4. Film taken April 2, 1936, again showing more tonicity of esophagus with thin stream of barium entering the stomach.

Auerbach's plexus was involved as the latter would still lack peristalsis despite diminished sphincter tonus.

CLINICAL COURSE

Cardiospasm occurs in individuals of all ages, the youngest being a child of two days reported by Jackson, while Walton[45] reports a case in a woman of seventy-three years. Aikman[1] reported 3 cases diagnosed by *x*-ray studies made on the third, fourth and fifth day of life. The average age is about forty years.[23] The ratio between females and males is as 3:1,[35] though Sturtevant[42] states that it is more common in males.

The main symptoms are substernal pain, dysphagia, vomiting of undigested food and loss of weight. The pain varies in severity from a mild discomfort to an agonizing pain so that in some cases the question of differentiation from a true angina confronts the clinician. Vomiting occurs shortly after eating and sometimes before the patient can leave the dinner table. No nausea accompanies the vomiting. In contradistinction to rigid, organic or cicatricial obstruction, liquids, especially cold ones cause almost as much difficulty in swallowing as solids. The weight loss progresses to a certain level and then as a rule the patient is able to maintain his weight at this level. The onset is frequently related to some psychic shock, such as the death of a member of the family. However, when once manifestations of cardiospasm have developed, there is no tendency to spontaneous remission. The condition usually does not rapidly progress to a fatal outcome even if untreated. Gould[12] reports a case which after duration of symptoms for forty-one years, finally succumbed to inanition.

The diagnosis is easily made by *x*-ray examination which reveals a dilated, elongated and atonic esophagus with the cardiac end of the esophagus constricted to an awl-like point. The diagnosis is confirmed by the use of the esophagoscope. Carcinoma and organic stricture must enter into the differential diagnosis. Cardiospasm, though more uncommon than carcinoma, is not as rare as is generally believed. MacMillan,[25] in a statistical study of diseases of the esophagus, found that carcinoma was the cause of 40 per cent of 878 cases of dysphagia, while cardiospasm was the etiological factor in 15 per cent.

TREATMENT

The accepted treatment at present is dilatation of the cardia. Numerous techniques have been developed of which the more common are the mercury bougies of Hurst,[15] hydrostatic dilators of Plummer,[33] air dilator of Tucker[43] and gastric tube and dilatation method of Einhorn.[6,7] The results of this method of treatment are best given by quoting the series reported from the Mayo clinic by Moersch[28] in 1932 treated by use of the Plummer dilator. Of 810 cases which had been observed, 804 had been treated; 670 patients were traced, 475 were considered cured, including those with slight intermittent dysphagia; 105 patients were moderately

relieved, 32 were slightly relieved and 32 received no benefit; 9 died from splitting of the esophagus, 2 died from starvation and 12 died at home, cause unknown. It is obvious that esophagoscopic manipulation is not the harmless procedure that one usually considers it. This is further supported by the reported mortality of 1.5 per cent by Mosher[31] in 938 esophagoscopies.

Walton[45] and Hill[14] have not obtained as uniformly gratifying results as Mayo's. When dilatation has not been successful some form of surgery has been employed. Mikulicz[27] first suggested gastrostomy with manual dilatation from below. Walton[45] reports a series of 14 cases treated in this manner with good results in 13; the other case died of cardiac failure. Judd[18] reports 3 patients who were improved by this method, but did not respond to Plummer's hydrostatic dilator.

Anastomosis between the esophagus and stomach has been attempted by Exner,[8] Gray Turner and others. This procedure is, however, exceedingly hazardous and should not be attempted unless other methods fail. Lambert[23] maintains that this is the method of choice where the esophagus has lengthened in addition to having become dilated and forms a sigmoid shaped curve with a portion of the curve lower than the cardia.

Heller[13] in 1914 suggested an operation analagous to Rammstedt's operation for congenital pyloric stenosis. He collected 16 cases in which the operation was performed, with good results in 12. This seems to be indicated only in those extremely rare cases where there is definite hypertrophy of the cardiac sphincter. In one case Watts[46] did a cardiplasty following the technique of the Finney pyloroplasty with good results. Freeman,[11] attributing the cause of the dysphagia to extreme lengthening, shortened the esophagus by intussuscepting the redundant length through a cervical incision. The patient has remained well for twenty-one years. Attempt has been made to correct the dilatation by employing plication (Reisinger[36]).

Knight's[19] experimental work suggested that improvement would result from sympathetic denervation of the cardia. To effect a denervation in the human, it is necessary to excise the left gastric artery with its surrounding fat and nervous tissue. This was done in 5 cases[20,21] with some improvement in all, though in one in which there was definite hypertrophy of the cardiac sphincter, there was the least amount of improvement. This denervation could also be accomplished by a bilateral cervicothoracic ganglionectomy designed to include the upper thoracic ganglia. One such case is reported from the Mayo Clinic[4] with relief of symptoms with, however, the accompanying Horner's syndrome. This does not seem warranted in view of the simpler surgical procedure as outlined by Knight.

With this résumé of the subject of cardiospasm, 2 cases are presented which were treated by sympathetic denervation as originated by Knight.

Case 1. M. R., female, aged fifty-seven years, was admitted to the Hospital of the University of Pennsylvania on January 8, 1935 with complaints of difficulty in swallowing, vomiting and upper abdominal pain. She had been in poor health for more than ten years. Approximately eleven years ago she had an attack of abdominal pain on the right side, with nausea and vomiting, for which an appendectomy was done. The patient, however, was told that the appendix was not "bad." At this time she weighed about 235 pounds. Throughout the next ten years she had attacks of upper abdominal pain of typical gall-bladder colic, which had increased in frequency and severity. In addition to this she vomited almost every evening for the past ten years. As soon as she would lie down she felt as if she were choking, had to cough and then vomit. Since December 3, 1934, the date of the death of her daughter, she had been unable to retain either liquids or solids and had lost weight rapidly and therefore was referred to the hospital. Physical examination revealed a fairly well nourished female, despite the loss of ninety pounds in weight. Otherwise the examination was essentially negative. *X*-ray examination showed obstruction at the cardia and a tentative

diagnosis of cardiospasm was made which was confirmed by esophagoscopic examination. Because of the definite hisotry of gall-bladder colic a laparotomy was done with the hope that the cardiospasm might be reflexly due to gall-bladder disease. At operation a calcified gall bladder containing stones was removed. The cardia was examined and by inverting the stomach wall over the finger the lower end of the esophagus was palpated but no constriction or mass could be demonstrated. Her convalescence was uneventful except for persistence of difficulty in swallowing solids though she was able to retain liquids. She was discharged on January 27, 1935, and readmitted on February 25, 1935. During the interval she had no abdominal pain but substernal pressure and a choking sensation which resulted in vomiting persisted. Ankle edema (nutritional) which had been noted on first admission had become more marked.

X-ray studies revealed the cardiospasm to be still present and esophagoscopy with dilatation was done on February 26, March 5 and 19, and April 6, 1935. In addition to the edema mentioned there were also signs of a peripheral neuritis and therefore she was given a high vitamin diet by stomach tube which was passed under direct vision, but the patient demanded removal of stomach tube. She appeared no better and refused to have further esophagoscopic dilatations. At this time she weighed 140 pounds. It was therefore decided to do a sympathetic denervation of the cardiac end of stomach and lower esophagus. On April 19, 1935, through an upper left rectus incision, the left gastric artery was resected with the surrounding tissue. Grossly this contained nerve filaments and was confirmed on microscopic section. Convalesence was uneventful and she was discharged on May 8, 1935. Since then she has been in the hospital in July and October, 1935 for check-up examinations. She has only vomited once since her denervation and in October had gained 30 pounds in weight. However, *x*-ray pictures still revealed a marked degree of achalasia and on one esophagoscopic examination a piece of orange pulp was found which she had eaten forty-eight hours previously. Her last follow-up examination was in May, 1936. At this time 10 per cent of the barium was in the stomach in half an hour, 25 per cent in one hour and no twenty-four hour residue. She weighed 195 pounds, which is 55 pounds above her lowest weight. Ankle edema and signs of peripheral neuritis had disappeared; in fact, she was symptom free.

Case II. A young, white female, aged nineteen years, developed normally to the age of sixteen years, at which time she weighed 143 pounds. In January, 1933, she began vomiting after taking food, before leaving the table, but if she could keep food down for half an hour usually did not vomit. This condition gradually became worse until July, 1933, when she was taken to another hospital where a diagnosis of tuberculosis of the lungs was made in addition to cardiospasm. She was allowed to return home unimproved and continued to loose weight dropping down to 84 pounds. She also began having night sweats. In September, 1933, she was admitted to Dr. Gabriel Tucker's service in the University Hospital where the diagnosis of cardiospasm was also made. In addition there was marked mottling of both lungs, especially the left, which was not typical of tuberculosis and which was taken to be an aspiration pneumonia. Of interest, was the history elicited that patient always slept on her left side and hence more spill into the left bronchus was shown by *x*-ray films and therefore more marked disease in left lung. Esophagoscopic examination confirmed the diagnosis and between September 26 and November 21, 1933, she had twelve esophagoscopies for treatment. She improved tremendously. Her lung lesion cleared remarkably and she gained 33 pounds in weight. However, at intervals of one to three months her symptoms would reappear and she would have to return for further dilatation, having sixteen treatments from December 15, 1933 to October 21, 1935. Because of the necessity of these frequent returns, it was decided to do a sympathectomy as was done in the first case. The operative procedure was done on October 14, 1935, without incident. Her immediate postoperative convalesence was without incident. However, she developed a choroiditis and following this a month after operation her pulmonary condition flared up and she ran an evening fever. Though symptoms of esophageal obstruction were less, there was still some dysphagia. Because of these residual symptoms, Dr. Gabriel Tucker dilated her cardia on two occasions, the last one before discharge being on January 21, 1936. She returned to her home and has been much improved up to the present time. She was

readmitted on April 1, 1936 for check-up. In this interval she has not vomited or regurgitated at all. She had no pain but complained of a slight soreness retrosternally when she ate. Esophagoscopic examination revealed no retention of food and a 8 mm. full lumen esophagoscope passed readily into the stomach. There was still *x*-ray evidence of cardiospasm and some residual pulmonary disease. In résumé some improvement was obtained by sympathectomy but the result has not been nearly as dramatic as in the first case.

DISCUSSION

Case 1 showed remarkable clinical improvement which could be attributed to the denervation alone. It is true that improvement was mainly clinical and *x*-ray examination still showed a dilated esophagus though the roentgenologic appearance shows definite improvement in tone. These *x*-ray findings were duplicated in second case. Plummer[33] states that only in a small percentage of cases benefitted by dilatation does the esophagus ever regain its normal size.

Of further interest in the first case was the ease with which the cardia could be explored by invaginating the stomach wall. No attempt at dilatation was made but this probably would be a worthwhile addition to the operative technique. Kümmel[22] and Gould[12] report cases where at least temporary benefit was obtained by manual dilatation without opening the stomach.

The improvement was not as marked in the second case. Here is emphasized the necessity of close cooperation between the endoscopist and the surgeon. Denervation made the dilatation by Dr. Gabriel Tucker easier without which, however, the case would have had to be considered a failure.

Case 11 also demonstrated a very interesting pulmonary complication, aspiration pneumonia, which had been diagnosed as pulmonary tuberculosis. No reference was made to this complication in the articles reviewed though Rice[38] incidentally mentions that one of his patients had been sent to a tuberculosis sanitarium. One would imagine that this complication is considerably more prevalent than perusal of the literature would tend to show.

CONCLUSIONS

1. Two cases of cardiospasm are presented which were benefitted by sympathetic denervation.
2. Attention is called to the aspiration pneumonia which may be a result of cardiospasm.
3. It is suggested that at the time of denervation it may be of value to dilate the cardiac sphincter by invaginating the stomach wall over the palpating hand.
4. Sympathetic denervation should be considered in all patients who have residual symptoms following dilatation.
5. Finally, these patients are most satisfactorily treated in a clinic with the combined efforts of internist, bronchoscopist and surgeon.

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Famous female imposters of the period (18th century) were Mrs. Mapp, a bonesetter, who was so successful that she could drive in from Epsom in a chariot and four, with gorgeously liveried servants, and Joanna Stevens, a widow, who, in 1739, actually succeeded in having her remedy for stone purchased pro bono publico by Act of Parliament. Her philanthropy went to the extent of agreeing to part with this valuable recipe for £5,000, but even a titled subscription list could not raise this sum in the first instance, and powerful influence was brought to bear upon Parliament, even Cheselden, Sharp, and Cæsar Hawkins vouching for her merits. The recipe was published in the London Gazette of June 19, 1739, and turned out to be a set of mixtures of egg shells, garden snails, swines' cresses, soap, and such vegetable ingredients as burdock seeds, hips, and haws. In each one of her certified "cures," the stone was found in the bladder after death.

Garrison's History of Medicine.

ABSCESS OF SPACE OF RETZIUS

REPORT OF CASE*

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PHILADELPHIA

ABSCESS of the space of Retzius is rarely encountered in gynecological practice, but if the occasional case is not recognized it might prove fatal. Early diagnosis and proper treatment, however, usually results in complete recovery.

The prevesical space was first described by the Swedish anatomist, Retzius, in 1856 and has since been called after him, the space of Retzius.[1] Although called a space it is not so in reality, being occupied by loose areolar tissue and the anterior and lateral lymph glands of the bladder. Its clinical importance is that it serves as an extraperitoneal approach to the urinary bladder and occasionally becomes the seat of a suppurative process.

Anatomically, it lies above the pubovesical ligament, partly in the pelvis and partly in the abdomen and is a component of the extraperitoneal space which extends from the pelvic floor to the umbilicus.[2] The space is bounded anteriorly by the symphysis pubis and the anterior layer of the transversalis fascia; posteriorly by the posterior layer of the same fascia, which passes down over the bladder to fuse with the visceral layer of the pelvic fascia, and the anterior border and inferiolateral surfaces of the bladder.[3] This space extends laterally as far as the hypogastric arteries and their visceral branches. It extends upward to the umbilicus between the peritoneum and the transversalis fascia where it is bounded on either side by the obliterated hypogastric arteries (lateral umbilical ligament). Below it is limited by the pubovesical ligament and the fascia covering the levator ani muscle.[4]

Some authorities refer to the umbilicovesical fascia, which arises at the umbilicus and extends downward to the anterior wall of the bladder where it joins the vesical fascia, and then proceeds downward to the pelvic floor where it unites with the pelvic fascia. Several centimeters below the fold of Douglas this fascia separates from the transversalis fascia, and it is between this layer and the transversalis fascia that the space of Retzius lies.[5]

The urachus is the remains of the fetal allantois and between the fourth and fifth month of fetal life, although still tubular, loses its lumen and becomes a solid cord.[6] The urachus in all probability plays a rôle in the extension of prevesical infections. It is extraperitoneal except in rare cases. The average adult urachus is 12 cm. long, 1.5 cm. wide and is usually adherent to the abdominal wall. Incomplete obliteration is the cause, in the newborn, of the escape of urine from the umbilicus. Pfeiffer[7] states that failure of the urachus to close at the vesical end is more frequent than persistent sinus at the upper end. He relates the observations of Wutz, who noted in 69 per cent of 74 cadavers of all ages, a funnel shaped indentation of the mucous membrane at the summit of the bladder through which a bristle could be passed into the lumen of the urachus. The influx of fluid during life is prevented, according to Wutz, by a transverse fold at the entrance of the urachus into the bladder. This gives rise to the possibility of bladder infections entering the urachus and spreading upward toward the umbilicus or by contiguity into the prevesical space. Binnie[6] cites a case in which a woman of twenty-nine years of age, with chronic cystitis of six years duration, complained of pain and tenderness in the hypogastrium. There was

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a line of induration between the bladder and the umbilicus and pus escaped from the umbilicus. There was a mass of granulation tissue at the umbilicus and through this a probe could be passed to the bladder. At operation the fistula led into a small diverticulum at the fundus of the bladder. A second case was that of a boy, nine years of age, with a tender hard tumor, 5 cm. in width, extending in the midline from the umbilicus to the pubis. Pus escaped from the umbilicus. A probe introduced into the umbilicus passed toward the fundus of the bladder for a distance of 4 cm. The sinus was between the peritoneum and muscular wall of the abdomen. Free drainage resulted in a cure.

Acute bladder conditions do not usually extend into the surrounding tissue. Chronic bladder infections, however, are more frequently the cause of pericystitis. Intravesical conditions of calculus, tumor, diverticula and tuberculosis lead to chronic cystitis which in turn may result in prevesical suppuration. Trauma and metastasis from some distant point of infection may result in an abscess of the space of Retzius. Goldberg[8] reports a case of prevesical abscess following acute mastoiditis in which the non-hemolytic streptococcus was obtained from both the mastoid and the prevesical abscess. This he considered conclusive proof of the role of the blood stream as the origin of the infection.

Extension of an intra-abdominal condition into the space of Retzius may occur. Pfeiffer[7] reports a case of tuberculous salpingitis and peritoneal tuberculosis which by adherence, ulceration and contiguity invaded the prevesical space. This was followed by cold abscess formation and extension upward to the umbilicus.

With the advance in pelvic surgery, abscess of the space of Retzius is infrequently encountered. In a few, however, after puerperal or other pelvic infections, the process may extend through the anterior abdominal wall and invade the prevesical and umbilical regions. Failure of recognition and inadequate treatment may result in the spread of the infection to the remaining extrafascial spaces in which a diffuse cellulitis may be set-up to be followed by septicemia and a fatal outcome.

The symptomatology of this condition is that of pain over the bladder when confined to the prevesical space. With extension toward the umbilicus, pain is a persistent complaint throughout the course of the infection. Bladder symptoms of dysuria and frequency are present until adequate drainage is instituted. Extraperitoneal localization is usually without gastrointestinal symptoms but spread to the retrovesical region is usually accompanied by nausea, vomiting, painful bowel function and either diarrhea or constipation.

The diagnosis is made on the finding of a suprapubic painful mass which is not reduced by catheterization. Later in the course of the condition with extension toward the umbilicus, one usually finds a mass in this region with protrusion and edema of the umbilicus. Early on pelvic examination a mass may be felt anterior to the bladder. Cystoscopic examination may show protrusion into the bladder at the site of the lower end of the urachus with inflammation of the overlying mucosa. The signs and symptoms of a septic process are present. Movement of the hip joints increases the pain so that the patient usually lies flat on his back with the thighs in flexion.[3]

Fortunately this condition is amenable to treatment. One method of approach is by transversely dividing the peritoneum, the anterior false ligament of the bladder and the posterior layer of the transversalis fascia just above the level of the vertex of the bladder, and then separating the bladder from the posterior wall of the symphysis by blunt dissection with the finger. The more common method, in the presence of infection, is by dividing the anterior abdominal wall down to and including the transversalis fascia, thereby allowing the escape of pus without contaminating the peritoneal cavity. In the following case the mass below the umbilicus was

incised and a drain inserted into the prevesical space.

CASE REPORT

M. G., female, colored, sixteen years of age was admitted to the gynecological ward, July 25, 1935, complaining of lower abdominal pain, vaginal discharge and dysuria since childbirth in February, 1935. The history relative to childbirth was negative; no previous pregnancies. Menstruation appeared at thirteen years of age, occurring regularly every twenty-eight days, of six days duration and without pain. Her last menses was the early part of May, 1935. Previous medical history was negative for any serious illness or operation.

Physical examination revealed undernourishment, temperature 100.3°, pulse rate 95 and respirations 20. There was no discernible focus of infection in the nose, mouth or throat. The thyroid was uniformly enlarged about twice the normal size but soft. The eyes were negative as were also the heart and lungs. Examination of the abdomen revealed an exquisitely tender mass, 7 cm. in diameter, rather firm in consistency, which extended one-half the distance from the symphysis pubis to the umbilicus. All other parts of the abdomen were negative to palpation. Pelvic examination disclosed the external genitalia to be negative. The cervix appeared normal but manipulation of it produced midline pelvic pain. The uterus was anterior, fixed and somewhat larger than normal, and anterior to it, in the region of the fundus, a mass could be felt which appeared to extend upward toward the umbilicus as well as anteriorly. The right adnexa was palpably enlarged and moderately tender. There was a moderate amount of vaginal discharge.

The sedimentation time on admission was 41 minutes. Examination of the blood showed 9.5 gms. of hemoglobin in 100 c.c., 4.05 erythrocytes, aniscocytosis and achromia; 10,700 leucocytes with 54 per cent neutrophiles. Urinalysis, except for a few clumps of pus cells, was negative. The Kline test for syphilis was negative.

The temperature showed a late afternoon rise of 100° to 104°F. Several days after admission an intramuscular injection of 10 c.c. of aolan was given and repeated every third day for four injections. On the eighth day after admission, Elliott treatments were started and continued daily for eleven days. After the second Elliott treatment the temperature varied between normal and 100°F. for about six days, only to rise again, but at no time higher than 100.3°F. On the twenty-sixth day after admission edema and swelling of the umbilicus appeared with a painful mass palpable and visible just below it. The following day, under ethylene, ether-oxygen anesthesia a posterior colpotomy was performed but no pus was obtained. An incision was made through the skin overlying the mass below the umbilicus and was immediately followed by the escape of a large quantity of greenish colored thick pus. A cigarette drain was inserted as far down as possible in the direction of the prevesical space. Convalescence was uneventful from this time except for an occasional elevation of temperature and drainage of pus at the point of incision below the umbilicus. At the time of discharge, thirty-six days after incision and drainage, pelvic examination revealed the uterus to be in anterior position, slightly larger than normal and somewhat fixed, but without the mass on its anterior wall as previously described. A painless firm mass, however, was still palpable in the right adnexal region. When the patient's dressings were changed, pus would escape through the incision on making pressure above the symphysis pubis.

Bacteriological examination of the pus disclosed gram negative, streptococcus types, many negative bacilli and many polynuclears, the final report being that of hemolytic streptococcus.

The sinus below the umbilicus continued draining for some months afterward, and occasionally pressure over the bladder area brought forth a spoonful or more of pus. At no time was there a febrile reaction and the patient was free from pain. On pelvic examination, however, the right adnexal mass was still palpable. This was thought to be inflammatory in origin for which the patient again was admitted for operation. This afforded an opportunity to investigate the pelvis to determine, if possible, the origin of the prevesical infection.

The patient was readmitted January 15, 1936, and except for the draining sinus below the umbilicus, which by this time had almost healed, was otherwise in good health. Operation the following day consisted of a median suprapubic incision with excision of the old scar and sinus. The omentum was densely adherent to the under surface of the abdominal wall and to the pelvic viscera. These adhesions were

freed and a right chronically infected tube and ovary were removed. There were a few rather coarse bands of adhesions extending from the posterior surface of the bladder to the anterior uterine wall. The space of Retzius proper was not visualized because of fear of contaminating the peritoneal cavity by any residual infection.

The patient made an uneventful recovery and was discharged on the thirteenth day. Several follow-up examinations in the dispensary disclosed the pelvis to be normal, the patient had gained weight and in general was markedly improved.

COMMENT

This case probably had its onset following childbirth, in spite of the normal puerperium. A latent childbirth infectious process appears to have been relighted which spread by way of the lymphatics to the prevesical space. Although cystoscopic examination was not done, the prompt amelioration of urinary symptoms following incision and drainage is against a chronic cystitis. The presence of a chronically infected right tube and ovary, and the absence of any other discernible focus of infection is in favor of a pelvic origin of the condition. However, one can only speculate, for there are many cases of pelvic inflammatory disease, and few cases of abscess of the space of Retzius.

SUMMARY

The anatomy of the space of Retzius is briefly considered. The rôle of the urachus in the dissemination of prevesical space infections is discussed; several cases are cited illustrative of this fact. Chronic bladder infections, intravesical conditions, trauma, metastasis and extension from intra-abdominal pathology may result in an abscess of the space of Retzius. The symptomatology and diagnosis are discussed. Early diagnosis and adequate drainage are stressed. A case report with fairly typical signs and symptoms is given.

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LEFT UROGENITAL MALDEVELOPMENT

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THE following case report concerns such an unusual number of maldevelopments that it seemed worthwhile to record it in the literature. References were found to many cases showing any one of the defects present here, but their association in combination was not duplicated, although closely approximated in a few instances.

The patient, H. P., a thirty-five year old female, was first seen on June 4, 1935, complaining at that time, of a painful lump in the left groin associated with nausea and vomiting for the past four hours. The diagnosis of left inguinal hernia, with incarceration was made and the patient was ordered to a hospital. She arrived at the West Baltimore General Hospital early in the morning of June 5, 1935, when the following history was obtained.

Family History. One sister is said to have a congenital deformity, making it impossible for her to have children. The exact nature of this deformity could not be determined.

Past History. Non-contributory.

Present Illness. For at least twelve years, the patient has noticed a lump in her left groin which would appear when standing and disappear upon lying down. Two months previously, the lump had become hard and tender and was associated with nausea and vomiting for twelve hours, after which time it reduced itself and the symptoms subsided. On June 4, 1935, the left groin again became hard, tender and swollen, following which nausea and vomiting appeared.

Physical Examination. General examination showed nothing of interest. Locally, there was a firm, tense, painful indurated mass in the left inguinal region, 20 by 20 cms. in size. During the examination the greater part of the mass was reduced, accompanied by some gurgling of gas. A small, freely movable lump remained at the external inguinal ring. An enema, administered at this time was effectual, being the first bowel movement since the onset of her illness. The blood and urine studies were normal. A rectal, but not pelvic, examination was made at this stage and no pelvic mass was noted. Inasmuch as the inguinal mass could not be entirely reduced, operation was decided upon, with a diagnosis of left inguinal hernia.

Under general anesthesia, a left oblique inguinal hernia incision was made, and the hernia sac readily identified. It was large, and when opened revealed an ovary and Fallopian tube, both discolored from their previous strangulation. A finger was inserted through the internal opening of the sac and, immediately encountered a large mass filling the left pelvis. Uncertain as to its nature a low midline incision was made. Inspection of the interior of the abdomen revealed a bicornuate uterus, in which the right cornu was fully developed, and apparently was accompanied by a normally situated right tube and ovary. The left cornu was underdeveloped, being smaller in diameter than an average tube. The left tube was still smaller in calibre and greatly elongated and extended down into the left inguinal canal. At its fimbriated end was a shiny, small, atrophic appearing ovary.

The right kidney was normal in its position, shape and size. No kidney could be felt in the left renal fossa. The mass, in the left pelvis, was retroperitoneal, movable and divided into two equal parts, the uppermost of which rose over the left iliac vessels, above the brim of the pelvis. The posterior peritoneum over the mass was incised and it was seen to be the left kidney. The division between the lobules, so far as one could tell by palpation and inspection, was complete, there being only a loose bridge of connective tissue joining the two. The ureter and vascular pedicle of the upper portion were plainly seen, as was the separate ureter of the lower segment for a short space at its hilus. Further dissection was not carried out to reveal the lower vascular pedicle, nor to determine whether the two left ureters fused or ran separate courses. The anomalous kidney was not disturbed, and the incised posterior peritoneum closed over it. The left atrophic uterine cornu, tube and ovary were excised and the left inguinal hernia repaired. Convalescence was uneventful during which a

subsequent pelvic examination revealed a normal appearing vagina and cervix, there being no evidence of division or maldevelopment of either.

seems to be solid without a lumen. There is a small ovary 1 × 2 × 3 cms., which is smooth and its external appearance resembles a testicle. On cut section there is seen marked cystic

FIG. 1. The drawing shows the atrophic cornu on the left with the underdeveloped tube and ovary extending into the hernial sac on that side. It also illustrates the arrangement of the pelvic kidney. The vascular pedicle of the upper lobe was readily demonstrated but that to the lower portion was not dissected.

The pathological report made by Dr. Ralph Mostwill is as follows:

Gross. The tissue consists of a rudimentary tube and another structure which also gives the appearance of a tube, but on cut section it

degeneration throughout the cortical area.

Microscopic. The ovary shows many dilated cysts lined by stratified low, columnar epithelium. The stroma shows marked hyalinization and fibrosis. There are few areas of normal

tissue left with very few normal follicles present.

Section from the cornu shows normal musculature, presumably uterine with many large blood vessels. There is no increase in fibrous tissue.

This report indicates that the patient had a maldevelopment of the left urogenital tract with a persistence of the canal of Nuck. Inguinal hernia in women is, of course, not rare, Watson[4] stating that 45 per cent of all hernias in females are inguinal. Also many cases of herniation of the tubes and ovaries have been collected. Heineck[5] compiled 137 such instances from the literature up to 1912. Of this series 5 had congenital anomalies of the genital tract, and one, in addition, had a pelvic kidney. In 1915 Rabinovitz[15] reported a thirty-five year old woman with bilateral ovarian inguinal hernia, in whom the uterus and vagina were lacking. Cullen[14] in 1910, recorded the case of a seventeen year old girl with each ovary in its corresponding inguinal hernia. She also had a right pelvic kidney, while the left kidney and ureter were lacking. Liesching[8] in 1929, wrote of a five year old child with her left tube and ovary strangulated in a left inguinal hernia while the left kidney was in the pelvis. The uterus and right ovary showed no developmental defects. Thus, in 140 cases of tuboovarian hernia, 8 showed deformities of the genital tract.

The occurrence of maldevelopments is much higher in cases where an uterus is found in the inguinal hernia sac. Watson[4] collected 56 such hernias, in 18 of which there were bicornuate uteri.

Cases of double and pelvic kidney have frequently been recorded. The latter occurred in Cullen's as well as Liesching's reports. Young and Davis[11] collected 26 instances of double kidney, to which Harpster[13] added 257. It is interesting to note in Girard's series of 44 cases of double kidney that hydronephrosis of that kidney occurred in 21, a high incidence of morbidity. The many variations of kidney and ureteral malformations are instructively illustrated by Pohlman,[12] Huntington[10] and Young.[1] Geraghty[9] reported 5 cases in which the lack of development revealed itself in the markedly diminished size of the kidney and ureter. Radasch[7] collected a number of instances where there was complete unilateral absence of the urinary tract. He found that in one-third of these females there was a bicornuate uterus. Guthrie and Wilson[6] wrote of a thirty year old woman in whom the right cornu of the uterus, tube, ovary, ureter and kidney were well developed while there was an entire absence of these structures on the left side. Wharton[2] pointed out the frequent associations of underdevelopment of the female genital tract with malformation of the urinary system, quoting 18 instances of ectopic kidney, 10 in males and 8 in females. Three of the males had genital deformities, and 2 of the females had gross uterine defects. He also mentioned 12 females in whom there was an absence of one kidney, and of these, 8 had deformed genital organs, an incidence of 66 per cent.

We collected from the records of the Union Memorial and Johns Hopkins Hospitals in Baltimore 45 cases of incomplete development of the female genital organs. Of these 2 had a left pelvic kidney and one an ovary in a right inguinal hernia.

SUMMARY

A case is presented which showed a marked congenital malformation of its genitourinary system, plus a persistence of the canal of Nuck on that side. So far as can be discovered, it is unique in the literature. A review of other articles on this subject indicates that disturbances in the development of the urinary tract in females are frequently associated with defects in the genital system; this is true particularly in cases of absent or ectopic kidney.

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Gastric syphilis is the lesion that most frequently simulates gastric carcinoma, both in its circumscribed and in its diffuse form, and its incidence is almost twice that of benign tumor, in our experience. Nevertheless, the fact must not be lost sight of that the gastric disturbances of syphilitic patients are rarely attributable to gastric syphilitic lesions, but to such conditions as neurosyphilis, functional disorders, gastritis, duodenal ulcer, and gastric carcinoma. The successful differentiation of a malignant lesion from a syphilitic one is often exceedingly difficult.

The Stomach and Duodenum by George B. Eusterman, M.D.
and Donald C. Balfour, M.D.

GYNECOMASTIA WITH CONCOMITANT TESTICULAR ATROPHY*

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GYNECOMASTIA, by no means a rare entity, has been an interesting subject of discussion, particularly in regard to the etiological factors involved. As early as 1849 Beau[1] observed the relationship between gynecomastia, pseudo-hermaphroditism and imperfect development of the sex organs. Some investigators sought to demonstrate this condition as a pathognomonic sign of a particular endocrine dyscrasia. Weber,[2] however, noted that enlargement of one or both male breasts may be an occasional result of a variety of disturbances, most clearly endocrine derangement, but the mammary enlargement should not be regarded as an absolute sign of disease of any one endocrine organ.

The literature bears many references to a varied etiology and includes spontaneous gynecomastia of puberty, as part of a syndrome associated with pseudo- and true hermaphroditism; postoperative development following prostatectomy;[3] testicular neoplasm (teratoma, chorioepithelioma, carcinoma);[4] hepatic cirrhosis; hyperthyroidism;[5] suprarenal, pineal and pituitary tumors;[6] testicular atrophy secondary to mumps; testicular atrophy secondary to trauma.[7]

The case presented is of interest owing to the concomitant relationship of testicular trauma and atrophy to the subsequent development of gynecomastia.

CASE REPORT

History. J. B., a nineteen year old, unmarried, white male, student, complained of pain in the left breast, unaccompanied by any swelling or tumor formation, in January, 1934. A month later he observed a small firm mass in the right breast and a similar nodule was soon observed in the left breast. The latter enlarged progressively until June, 1934, while the right breast progressed in size until September, 1934. Both breasts remained tender to palpation, although pain per se had ceased by September of the same year. At no time was there any discharge from either nipple.

FIG. 1. Appearance of patient on admission.

The past history related an injury at the age of nine years, at which time the patient was struck in the left testicle while at play. No orchitis developed subsequent to this initial injury. At the age of fourteen years while playing hockey he was struck in the left testicle by a forcibly hit puck. Following this injury he was confined to bed for ten days with a severe bilateral orchitis. The swelling gradually subsided but for several weeks thereafter the patient remained weak. He noticed that the left testicle remained smaller than the right one. In April, 1933, at the age of sixteen years, following a slight injury to the right testicle during convalescence from typhoid fever, he suffered another attack of orchitis. At this time the left testicle appeared to be markedly atrophied and a diminution in the size of the right

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testicle was also noted. In July, 1933, there was a third and comparatively mild attack of orchitis. Some time later the onset of the breast symptoms was noted.

tially negative except for a slight tendency toward increased adipose tissue about the hips and breasts, but the hair distribution was normal for a male of his age. The breasts were

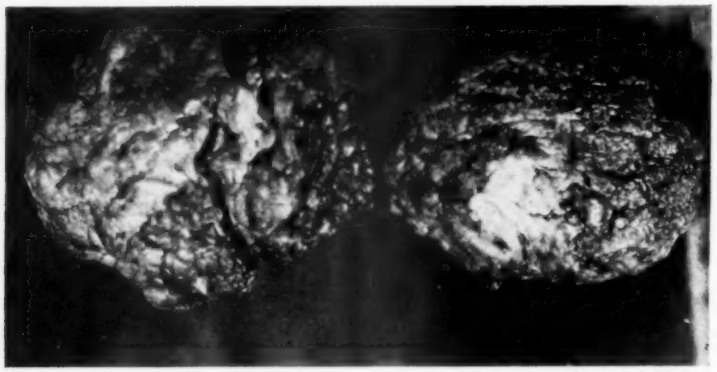

Fig. 2. Gross specimens showing uneven lobulated surfaces, due to the irregular fibrous ramifications through the fatty tissue, the larger gross specimen being that of the right breast.

Fig. 3. Right breast. Dilated mammary gland ducts and scattered acini embedded in fibrous tissue and fat (medium high power).

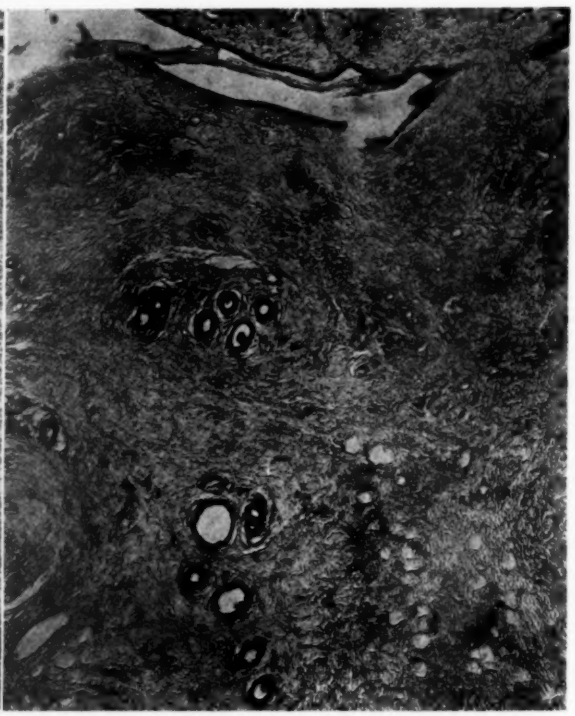

Fig. 4. Left breast. Some of the ducts contain coagulated albumin and exfoliated epithelium (medium high power).

The family history was irrelevant; there were no siblings and no familial history of excessive breast hypertrophy or related endocrine dyscrasia could be ascertained.

Examination. The patient gave the appearance of a fairly well developed normal male, 6 feet in height, about twenty years of age, and weighing 175 pounds. Examination was essen-

both enlarged and appeared to be the size and configuration of those of a well developed adolescent female, resembling normal female hypertrophy at puberty. The nipples were rather prominent and surrounded by a slight degree of areolar pigmentation. The left breast measured six inches in its transverse diameter and five inches in its vertical diameter. The

right breast was six and one-half inches by five inches, respectively. The areolae of both breasts measured one and one-quarter inches transversely and one and one-sixteenth inches vertically.

No discharge could be expressed from either nipple nor were any axillary nodes palpable but the breasts, however, were tender. The only other significant findings on general examination were the almost complete atrophy of the left and partial atrophy of the right testicle. The latter was about three-fourths the normal size but of normal consistency and was associated with a mild varicocoele. The remnant of the atrophied left testicle was nothing more than a very small fibrotic nodule in the left side of the scrotal sac.

The Kahn and Wassermann tests were negative. Urinalysis showed no sugar, albumin or pathological sediment. *X*-ray picture of the skull on July 16, 1934, was reported as showing no gross abnormality and the sella turcica was described as being of normal proportional dimensions with no evidence of erosion or pressure atrophy. The basal metabolic rate on July 18, 1934, was 4 per cent below the average normal.

Course and Operation. Glandular extracts by mouth and twenty-three injections of hormone-like substances hypodermically were given from August to October, 1934, without any apparent effect in arresting the mammary development. The patient was admitted for surgery, and bilateral subcutaneous ablation of breast tissue was effected through "Warren-Thomas" incisions, care being taken to preserve the nipples and areolae. The gross specimens are shown in Figure 2.

Histologically, typical sections of the right and left breast, respectively, are shown in Figures 3 and 4.

The pathologic report by Dr. L. H. Meeker of the New York Post-Graduate Hospital Columbia University, described "a stroma of hyaline fibrous tissue in which there are widely spaced tubules lined by one to several layers of cuboidal epithelium. In part the tubules represent gland ducts and are somewhat dilated. In part the fibrous tissue contains widely spaced gland acini which extend irregularly into the surrounding fat. In the stroma beneath the epithelium there is a mild lymphocytic infiltration.

"*Diagnosis:* Gynecomastia, bilateral."

COMMENT

The case described is of particular interest in view of the extensive research being done in connection with the various hormones and their relationship to the development of physiological and pathological breast conditions. The inhibitory action of testis hormone on breast development is, however, still not definitely established. Kriss[4] tried to show by a study of selected cases that no distinct relationship existed between "cessation of testicular inhibitory action" and mammary gland hypertrophy. While there is lack of conclusive evidence in this case that the gynecomastia developed as a result of testicular deficiency there was nevertheless a concomitant finding of gynecomastia and testicular atrophy.

SUMMARY

1. The case presented is clinically and histologically one of true bilateral gynecomastia in a well developed nineteen year old white male.

2. The history relates repeated testicular trauma with subsequent atrophy of the left testicle.

3. Mammary enlargement comparable to female puberty hypertrophy developed concomitant with testicular atrophy due to trauma.

4. The mammary change and testicular alteration were not demonstrably influenced by endocrine therapy administered hypodermically and by mouth.

5. Total subcutaneous ablation of both breasts with preservation of the nipples and areola was accomplished by surgery.

6. Although the gynecomastia was associated with testicular atrophy, there has been no conclusive demonstration that testicular hormone inhibits mammary gland development.

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Long before the days of ether, surgeons began to look beyond mere speed in amputating; and the character of the stump as regarded in future usefulness was most carefully considered by Cooper. He followed the teaching of his prodecessors in making long skin flaps, saying, "Don't cover the stump with muscles. They will retract and draw back the skin. Use skin only." He dressed the stumps with a light alcohol compress, and applied a tight roller bandage to immobilize the muscles. He seems to have frequently expected primary unions, for he directs that the dressings should not be disturbed until the eighth day, and that, pending that dressing, the temperature of the two limbs should be compared daily by the surface thermometer.

Surgical Memoirs by James Gregory Mumford, M.D.

MYOSARCOMA OF STOMACH

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SARCOMATA of the stomach are exceedingly rare, comprising only about 1 per cent of gastric malignancies.

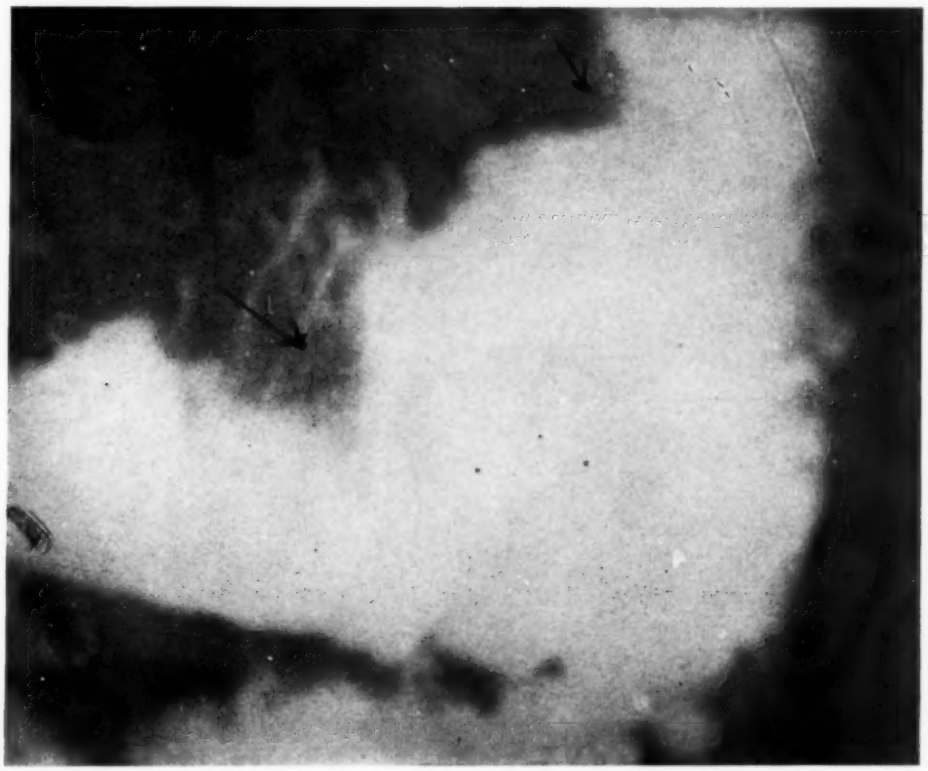

FIG. 1. Large ulcerating lesion on the lesser curvature near the pylorus.

Myosarcoma is difficult to distinguish from spindle cell and round cell sarcoma, though the latter are encountered more frequently. Ewing[1] says that about 1 per cent of gastric malignancies are sarcomata while Fenwick[2] places the percentage a little higher.

The etiology, pathology and clinical aspects of myosarcoma have been discussed adequately by Gaber, Tufts and Witte[3] and Edwards and Wright[4] have reviewed the literature up to 1933 and found reports of 38 cases. Gaber, Tufts and Witte[3] report one case with recovery following operation.

Myosarcomata of the stomach usually arise from the curvatures and often project into the peritoneum as solitary solid tumors and cystic tumors and may attain enormous size. The "exogastric" forms appear usually as sharply defined tumors and sometimes have a distinct pedicle. The "endogastric" forms are usually also pedunculated.

The structure of the myosarcoma varies; sometimes the cells are distinctly muscular in type, while in other cases the predominant cell may be of the large spindle cell variety. Sometimes there is a mixture of cells.

The following case is reported because of the rarity of the tumor and also because of the few symptoms which the patient presented, namely, marked anemia and slight indigestion.

J. G. J., aged thirty-one years, American, was admitted to the Memorial Hospital on February 3, 1936, complaining of pallor, fatigue and indigestion. The present illness dates back ten

years when the patient became conscious of increasing pallor. This was the only symptom noticed until about two months ago when the patient noticed acute pains in the epigastrum following meals. The history is interesting due to the fact that it is almost entirely negative except for the increasing pallor and slight fatigue on exertion. The appetite has always been good. The bowels moved regularly twice daily. There had been no loss in weight, vomiting, hematemesis or melena.

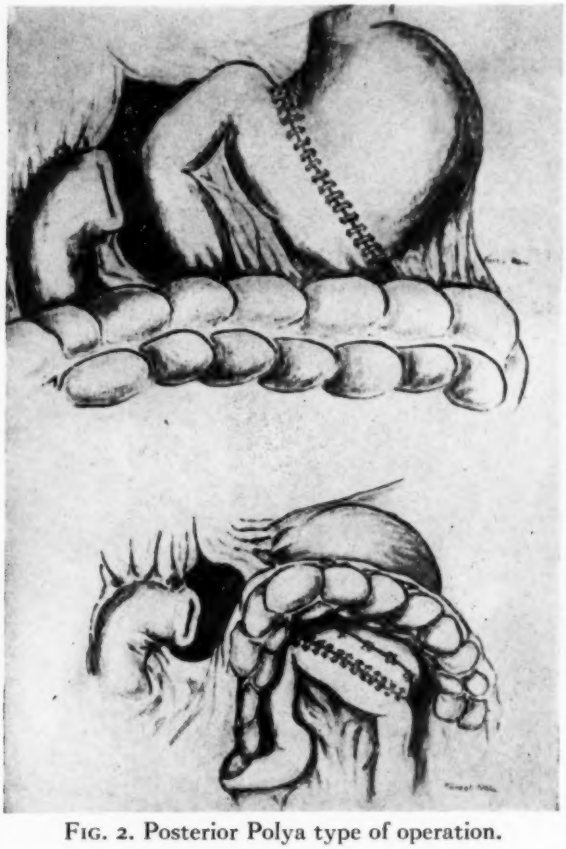

Fig. 2. Posterior Polya type of operation.

The past history was negative except for the usual childhood diseases and malaria in 1920. There were no previous operations. The family history was essentially negative.

Physical examination revealed a well developed white male of about the stated age of thirty-one years. There was a marked pallor of the skin. The physical examination was negative except for a marked pallor. The blood pressure reading was 110 systolic and 70 diastolic. There was slight tenderness on pressure in the epigastrum but no rigidity, masses nor scars. On admission the red blood cells numbered 1,390,000 and the hemoglobin was 35 per cent. There was a marked achromia, a moderate anisocytosis and an occasional poikilocyte. The white blood cells numbered 7550; 75 per cent of which were polymorphonuclears, 1 per cent were basophiles, 26 per cent were small lymphocytes and 5 per cent were metamyelocytes. The urea nitrogen was 18 mgs. per 100 c.c. The blood chlorides were .537 per cent. The urine examination was negative.

Fig. 3. Resected specimen consists of about four-fifths of the stomach and shows a large calloused, malignant ulceration.

The *x*-ray revealed a large ulcerating lesion on the lesser curvature of the stomach near the pylorus (Fig. 1).

Four transfusions of whole blood by the multiple syringe method were given on four successive days, totaling 2310 c.c. The red blood count following these transfusions was 4,000,000 with 74 per cent hemoglobin.

The abdomen was opened on February 7, 1936, under ethylene and ether anesthesia, through an upper left rectus incision. On examination of the stomach a large ulcerating growth was found on the lesser curvature of the stomach near the pylorus with evidence of glandular involvement in the immediate area. There was an attachment to the pancreas so that resection of the pylorus had to be more extensive than usual. About four-fifths of the stomach was removed and a posterior Polya anastomosis was done, joining the side of the jejunum to the end of the stomach (Fig. 2).

Grossly, (Fig. 3), there was a large, dollar sized crater lesion on the lesser curvature of the stomach about 2 cm. from the pylorus. The walls of the lesion were markedly raised and considerably infiltrated. Opposite the base of the crater there were several large glands forming a conglomerate-like mass about 4 cm. in diameter.

Fig. 4. Photomicograph shows the myosarcomatous nature of the growth.

Microscopically, (Fig. 4), the entire wall of the stomach and the glandular mass were made up of densely packed, very large spindle shaped cells having fairly long fibrils. There were a number of mytotic figures. No epithelial cells were found. A pathological diagnosis of myosarcoma, grade IV malignancy, was made.

The patient made an uneventful recovery and was dismissed from the hospital February 18, 1936, fifteen days following admission and eleven days following operation. At the present time the patient is in good health and has returned to his work in the oil fields.

CONCLUSION

A case of myosarcoma of the stomach is reported. There were practically no symptoms except those from a secondary anemia. Any case with an unexplained anemia should have a fluoroscopic examination made of the stomach to exclude ulcerating or polypoid lesions. Lesions of the right colon will likewise cause a severe secondary anemia.

The adequacy of radical resection for malignancies in the stomach has been shown a number of times. We are hopeful that this patient will be cured, however, if palliation only has been afforded it will be quite worthwhile.

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RIGHT PARADUODENAL HERNIA

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RETROPERITONEAL hernia is a relatively rare condition. While there are several types of hernia which occur behind the posterior parietal peritoneum, right and left paraduodenal hernias are the two types of chief importance. Forty cases of right paraduodenal hernia have been reported in the literature since Klob gave the first description of this condition in 1861. There have been more than twice this number of left paraduodenal hernias reported. The following discussion, however, will concern itself principally with right paraduodenal hernia and the report of an additional case of this condition.

Exner in 1933 classified the 40 cases of right paraduodenal hernia by considering these cases in three periods; from 1861 to 1903, from 1904 to 1923 and from 1924 to 1933 inclusive. He made a comparison of the number of cases described with the number of cases operated, the number which recovered after operation and the number diagnosed preoperatively in each of these three periods.

Period	Cases Reported	Cases Operated	Cases Recovered	Diagnosed Correctly
1861–1903	19	4	1	0
1904–1923	10	8	1	0
1924–1933	11	11	9	2
Totals.....	40	23	11	2

It is interesting to note how few cases were operated in the first group. There was only a single recovery in each of the first two groups, making a total of only 11 reported recoveries from this condition since it was first described in 1861. It is only in the last group that a correct diagnosis was made of the condition before laparotomy.

ETIOLOGY

Folds of peritoneum forming small fossa at various points about the terminal duodenum have been noted at necropsy and at the operating table from time to time. It is thought that these folds and the resulting fossa are formed in the development of the intestinal tract as early as the fourth month of intrauterine life.

These fossa when present vary in size and location. One of these, the fossa of Waldeyer, is the site where right paraduodenal hernia is formed. The most common position of the fossa of Waldeyer is in the beginning mesojejunum, just behind the superior mesenteric artery and immediately below the duodenum.

There are several theories as to the origin of retroperitoneal hernia:

1. Treitz contended that a loop of small intestine insinuated itself in one of these peritoneal fossa and due to intestinal pressure, the fossa became progressively enlarged until a retroperitoneal hernia was formed.

2. Andrews claimed that this did not take place but that retroperitoneal hernias were formed due to the imprisonment of small bowel by the mesentery of the developing colon.

It seems that there are some arguments which give weight to the first theory and others which support the second.

Andrews felt that the theory of Treitz was wrong because:

1. Differences in pressure are entirely lacking within the abdomen, so that intestine could not insinuate itself in one of the peritoneal fossa already present and enlarge it to form a hernial sac.

2. Many similar fossa are located in various parts of the peritoneal cavity which are not affected by hernia.

3. In all but a few cases the hernias have been either total or subtotal.

4. Small gut only is contained in the hernial sac; omentum is never found.

5. There was a case reported by Vogt of one type of retroperitoneal hernia occurring in a newborn infant.

There are several points which appear to be in disagreement with the Andrews theory:

1. Peristalsis of a loop of small intestine could account for its being insinuated in one of these fossa, and the enlargement of the fossa to form a sac, without any difference in intra-abdominal pressure occurring.

2. Brown reported a case of retroperitoneal hernia in which the sac was anterior to the ascending colon.

ANATOMICAL CONSIDERATIONS

Right paraduodenal hernia may be differentiated from left paraduodenal hernia as follows:

1. In right paraduodenal hernias the opening of the sac is on its left side and faces left, while in left paraduodenal hernias the opening of the sac is on its right side and faces right.

2. In right paraduodenal hernias the superior mesenteric artery runs in the neck of the sac anteriorly, while in left paraduodenal hernias the inferior mesenteric vein runs in the neck of the sac anteriorly.

PATHOLOGY

The most frequent pathological finding is that of intestinal obstruction, either of the acute, subacute or chronic types. Marked intestinal dilatation and hypertrophy of the intestinal wall has been found in some cases of the long standing chronic types.

Some retroperitoneal hernias have been discovered at necropsy, without evidence of intestinal obstruction either during life or at the postmortem table.

DIAGNOSIS

1. The clinical picture present in the majority of the 40 cases of right paraduodenal hernia was that of intestinal obstruction. In some it was that of acute obstruction; in others it was that of a chronic or recurring type of strangulation. Vomiting, however, was not as prominent a symptom as in most cases of obstruction due to other causes. When present, it has usually consisted of gastric contents, and later some bile stained mucus, rather than any fecal vomitus. This is only natural because the point of obstruction in the greater number of cases has been high up in the gastrointestinal tract. Moynihan believed that visible peristalsis and a palpable resonant, gurgling, balloon-like tumor in the lower right abdomen, later involving the whole abdomen, were of significance in diagnosing the condition.

2. The presence of a tympanitic mass must not necessarily be present, as the intestines in the sac may be collapsed, as they were in the case reported in this paper.

3. *X*-ray pictures may be helpful in making a diagnosis. Exner says that the clumped appearances of the intestinal coils, as if they were contained in a bag, is the most characteristic sign of right paraduodenal hernia.

4. A few cases have remained symptomless throughout life and have been discovered only at necropsy.

5. Since there are reports of only 40 authentic cases of right paraduodenal hernia in the literature and most of these are single case reports, it is not surprising that the diagnosis has only been made twice before operation or necropsy; once by Taylor reported in 1930 and the second time by Exner who made his report in 1933. Indeed, one is fortunate to have seen a case of right paraduodenal hernia in a lifetime.

TREATMENT

Paraduodenal hernias are treated surgically. The hernia must be reduced and in so doing, the relationship of vessels to the neck of the sac should be ascertained so as to avoid injuring them during the process. The sac should be obliterated when possible and edges of peritoneum sutured together. Cases where the blood supply to the bowel has been impaired are given the same treat-

ment as they would receive in other types of obstruction.

CASE REPORT

On December 31, 1932, a boy, fourteen years of age, was admitted to the Youngstown Hospital complaining of abdominal pain. The patient stated that early that same morning he had been seized with a sudden attack of violent cramp-like abdominal pain which "doubled him up." About thirty minutes after this he became nauseated and began to vomit. The pain, nausea and vomiting persisted. Following these symptoms there had been a bowel movement which was apparently normal.

The patient had previously enjoyed good health and outside of the usual diseases of childhood had had no other illnesses or symptoms of any kind. There was no history of any operative procedures or injuries.

Examination revealed no pathological findings except in the abdomen. The patient was lying in bed with both thighs flexed. Tenderness was present on palpation over the whole abdomen, especially over the lower right quadrant. Both recti muscles were spastic, the lower left rectus in particular. No palpable masses and no evidence of fluid were present in the abdomen. No marked distention was present. Rebound reflex was absent. The urine was negative. A blood count showed a moderate leucocytosis.

The patient was examined by several members of the surgical staff and it was the general concensus of opinion that this was an atypical case of acute appendicitis or a case of partial intestinal obstruction, with etiology unknown.

The afternoon of the same day the boy was operated. Through a right pararectus incision, and after mobilizing the cecum, a retrocecal appendix was removed. The gross pathological appearance of the appendix did not account for the clinical picture of the patient before operation.

Upon further exploration of the abdominal cavity, a sac composed of peritoneum was encountered medial to the cecum and dorsal to the omentum. This sac, which was about the size of a very large cantaloupe, contained almost all of the small intestine. The outlines of the intestinal loops could be seen vaguely through the semitransparent sac which was free on its anterior aspect. The small intestine entered this sac through an opening in its upper left side, just beneath the terminal duodenum.

It was only then that I realized that we were dealing with a retroperitoneal hernia, a condition which I had previously heard described, but which I had never seen. The exact relationship of the superior mesenteric artery to the neck of the sac was not noted, as I was unaware of its anatomical significance at the time of operation.

The inferior margin of the neck of the sac was split and the loops of small intestine were milked out of the sac. The intestine was in a collapsed condition and showed no sign of impairment to its circulation. The redundant portion of the sac was excised and the remaining edges were approximated by a suture, obliterating the retroperitoneal sac which originally contained small gut. Closure was then made without drainage.

The boy had an uneventful convalescence and on the eleventh postoperative day was dismissed from the hospital.

SUMMARY

1. This case of right paraduodenal hernia, as far as could be determined, was the forty-first to be reported in the literature, the twenty-fourth to have been operated and the twelfth to have recovered.

2. Single case reports have been in the majority.

3. The number of cases of right paraduodenal hernia diagnosed correctly before laparotomy still stands at two; the same as reported by Exner in 1933.

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ERYSIPELAS COMPLICATING LABOR AT TERM

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ERYSIPELAS is a relatively infrequent complication of labor, occurring more often as a postpartum infection. The literature contains isolated case reports, as cited by Middleton,[1] and does not offer any accumulated statistical data referable to the condition as a complication of pregnancy. Therefore, we are dependent on individual experiences for information and enlightenment regarding this condition.

Ordinarily, erysipelas carries a general mortality of 16.4 (±) 3.6 per cent, as recently reported by Keefer and Spink.[2] We may assume that this figure will be even greater when erysipelas is superimposed upon pregnancy, inasmuch as Williams[3] states that any septic condition offers a worse prognosis in pregnancy than at other times. In addition, the susceptibility to puerperal sepsis is markedly increased should labor ensue during the course of erysipelas. Fortunately, however, many cases of erysipelas that occur in expectant mothers subside before labor intervenes, thereby precluding this eventuality. Also, as mentioned by DeLee,[4] the seriousness of this complication varies with the anatomical location of the erysipelas lesion, with the danger increasing as the lesion approaches the genital region. In these cases we are concerned not only with the potential dangers to the mother, but also the hazards to which the baby is subjected. Reporting on this phase of the subject, Ottow[5] cites the cases of Kaltenbach, Runge, Stratz, and Lebedeff as examples of probable transmission of erysipelas from mother to child during pregnancy. Ottow himself cites a similar case, but concludes that there is no intra-uterine transmission of erysipelas from mother to child. He feels that only a toxic cutaneous manifestation can be conceded in these cases which has been interpreted as intra-uterine transmission.

The treatment of erysipelas during pregnancy is essentially the same as that of erysipelas generally. The various therapeutic measures employed include cold applications of a saturated solution of magnesium sulphate, collodion, antitoxin, *x*-ray, quartz light, and blood transfusion. The use of erysipelas antitoxin during pregnancy is not particularly encouraged by Hoyne.[6] Should labor set in, one should be prepared to carry out the extreme aseptic care required during a delivery of this type. This can be readily appreciated without further comment. One must also guard the baby by its immediate isolation from the mother, and in maternities the problem of nursing care and isolation for the protection of other parturients cannot be too strongly emphasized.

The relative infrequency of this complication, together with the many interesting phases, prompts the reporting of the following case.

Mrs. G., white, aged twenty-five years, American, married, was admitted to the Prospect Heights Hospital, February 2, 1936, after having been seen in consultation at home. Her family history was entirely irrelevant. Her past personal history showed measles during childhood, and a tonsillectomy in 1934. She was married two and a half years; her husband was in good health. Menstruation started at the age of fourteen years, occurring regularly every twenty-eight days, of four days duration, and without pain or clots. The last menstrual period started May 11, 1935. There were no previous pregnancies.

Present Illness. The patient had an uneventful pregnancy up to two weeks prior to hospitalization, at which time a sore throat developed; a few days following this a swelling appeared over the left side of the neck which

subsided in several days. Three days before admission, redness and swelling of the face and right leg appeared which continued to spread slowly. At that time her physician incised an abscess of the right nostril and obtained one ounce of greenish-yellow pus. She was given an injection of erysipelas antitoxin the day prior to her hospitalization. Weakness, fever, headache, chills, sore throat, obstructed breathing and constipation were the outstanding symptoms. Labor started at 4.00 A.M. the day of admission.

Physical Examination. The temperature was 101°; pulse rate 140, respirations 22, and blood pressure, 140/90. The pertinent findings were: A typical erysipelas lesion involving the nose and both cheeks, spreading over the angle of the jaw on the right side, with moderate cervical, postcervical, and submaxillary glandular enlargement, confined to the right side; throat congested; heart normal except for a rapid rate; no abnormal lung findings. The left lower extremity was essentially negative, but the right presented a typical erysipelas lesion, with the skin red, tense and glistening. The lesion involved the entire leg, and was marked by a vesiculation of its upper third. The abdomen showed a full term pregnancy with the fetus in R. O. A. position, and the head dipping into the brim. No fetal heart could be heard at the time. Pains were fairly strong and were recurring at five minute intervals. Rectal examination revealed a three-and-a-half finger cervix, with moderate effacement, and the head well engaged.

Progress of Labor. The patient was tired and appeared acutely ill. Morphine sulphate, gr. ¼, was given and the patient's general condition improved during the next few hours. Erysipelas antitoxin was administered during labor. Complete dilatation with the head on the perineum was noted on rectal examination at 10.00 P.M., eighteen hours after the onset of labor. At this time the pulse became rapid and thready, with cyanosis and circumoral pallor developing. Digifoline, morphine and atropine were administered. At 10.30 P.M., under nitrous oxide-oxygen-ether anesthesia the delivery was terminated by episiotomy and low forceps as an emergency procedure, in the interest of the mother. Fifteen minutes later the placenta was expressed intact, with about a 100 c.c. blood loss. The episiotomy wound was sutured with interrupted No. 2 chromic catgut and after the intravenous administration of 500 c.c. of a 10 per cent glucose solution, the patient's general condition was greatly improved. The delivery was accomplished in bed, in an isolation suite on the surgical floor.

Puerperium. The puerperium, immediate and remote, was marked by several interesting features until the patient's discharge from the hospital March 29, 1936, fifty-six days following delivery, and may be summarized as follows:

The fundus involuted fairly well, and reached the level of the symphysis at the end of two weeks. Uterine bogginess, associated with slightly foul lochia, responded promptly to the use of gynergen during the second week.

The episiotomy broke down at the end of a week, sloughing at first, but it granulated well and was clean at time of discharge from the hospital. The perineum was sprayed with 4 per cent mercurochrome daily for the first two weeks. Following this the wound surfaces were painted with a 10 per cent silver nitrate solution.

A serum reaction appeared one week postpartum, characterized by large weals, accompanied by intense itching which persisted for thirty-six hours.

The lesion on the face cleared in a few days, but the right cervical, postcervical and submaxillary glands, and the left cervical glands suppurated at various stages of the convalescence, necessitating incision and drainage for relief. A retropharyngeal abscess was incised one day postpartum, with the liberation of about one-half ounce of greenish-yellow pus.

The right leg became quite edematous, with the erysipelas lesion extending to the midthigh. Desquamation of the skin covering this area appeared on the eighth day after delivery. On the tenth day fluctuation developed within the confines of the lower extremity lesion. Large quantities of pus were liberated from this area, following multiple incisions on February 16 under nitrous oxide-oxygen-ether anesthesia. A continuous subfascial pus cavity was found to communicate from the right midthigh to the right ankle. This cavity was packed with gauze, and Penrose drains were introduced. Dakin irrigation was later instituted, and finally irrigation with merthiolate solution was utilized. The edema gradually subsided and the infection cleared. The wounds all healed prior to discharge from the hospital, with the exception of a lateral right leg incision.

The temperature course was septic, reaching its peak of 104.6 on February 17, flattening out by March 6, and continuing normal from then until the date of discharge.

The pulse rate during delivery was rapid, over 140, and thready. After delivery it ranged between 90 and 120 for three weeks, except on February 18 and February 23, when it reached 130 and 140 respectively. From February 24 on it remained between 80 and 100.

Therapy. The patient received two blood transfusions. On February 17, 200 c.c. of citrated blood were administered, and on March 13 she was transfused with 250 c.c. of citrated blood.

The other forms of therapy included sedatives, oxytoxics, antitoxin, intravenous glucose, liver and iron, digitalis, and surgical intervention as the occasion warranted.

The patient was out of bed March 24, and discharged March 29, fifty-six days following admission to the hospital. Her general condition was good, and all wounds were healed except the episiotomy wound and the leg wound mentioned. The former was clean and granulating.

A menstrual period occurred March 21 and lasted two days. Pelvic examination was done at the office June 13, and disclosed that the pelvic floor was healed, the cervix was slightly eroded, the uterus was well involuted and in normal position; the adnexa were grossly normal.

The baby was a female and weighed 6 pounds, 9 ounces at birth. She appeared perfectly normal in all respects, and was immediately isolated from the mother. No evidence of any skin lesion developed at any time up to her discharge from the hospital, March 16. The discharge weight was 8 pounds, 2 ounces. The cord separated normally nine days after birth, leaving a clean, moist surface which healed without infection. The baby was kept on a formula, with viosterol and orange juice added at the end of the second week.

Urinalyses (Catheterized Specimens). Feb. 3, 1936. S. G. 1.020; acid; trace of albumin and sugar; 3 to 5 white blood cells per high power field, rare clump.

March 3, 1936. S. G. 1.010; acid; no albumin or sugar; rare leucocyte.

Blood Cultures. Feb. 3, 1936; no growth reported on Feb. 4, Feb. 6, and Feb. 10, 1936. Feb. 29, 1936; no growth reported on March 2, and March 5, 1936.

Roentgenogram. Feb. 17, 1936; no bone involvement of right leg.

Pus Cultures. Feb. 3, 1936; retropharyngeal abscess: Hemolytic streptococci and staphylococcus albus.

Feb. 16, 1936. Right leg: Pure hemolytic streptococci. Cervical glands: Pure hemolytic streptococci.

LABORATORY DATA

BLOOD COUNTS

1936	Red Blood Cells	Hemoglobin, Per Cent	Color Index	White Blood Cells	Polymorphonuclears, Per Cent	Eosinophiles, Per Cent	Basophiles, Per Cent	Lymphocytes, Per Cent		Monocytes, Per Cent
								Small	Large	
Feb. 3.....	4,340,000	75	0.9	31,850	74	0	0	13	10	3
Feb. 7.....	3,320,000	52	0.8	11,800	86	0	2	8	1	3
Feb. 13.....	3,610,000	58	0.9	12,550	75	0	1	17	6	1
Feb. 20.....	3,520,000	63	0.9	9,250	67	1	0	30	1	1
March 10....	3,610,000	60	0.8	10,650	60	0	0	37	0	3

SUMMARY

A case of erysipelas complicating the labor of a full term pregnancy is presented.

Episiotomy and low forceps were required to terminate labor as an emergency procedure in the interest of the mother.

A hemolytic streptococcus was cultured from all suppurating areas, in the retropharynx, cervical glands, right leg and thigh.

A living baby was born without any manifestation of erysipelas, and was discharged from the hospital in good health.

The mother was discharged from the hospital fifty-six days following admission, fully recovered except for a granulating perineum and an unhealed incision of the right leg.

DISCUSSION

The outstanding obstetrical problem confronted in this case was the challenge of a virulent streptococcic infection to the demanded emergency operative intervention. Due to an overwhelming infection, the parturient presented a critical picture during the perineal stage. The obstetrical procedure of episiotomy and low forceps to terminate labor ordinarily required to meet such an emergency, was very evident. Yet, in the presence of a complicating erysipelas, hesitancy was experienced in risking the possibility of an almost certain postpartum infection. The outcome in this case justified the obstetrical procedure.

This is only a single instance and no conclusions are warranted. However, this case should at least encourage the employment of emergency obstetrical procedures in similar situations, under proper working facilities.

The additional problem of importance is that of proper isolation for the protection of the mother, the baby and other parturients. This can only be met, as it was in this case, by the cooperative efforts of the nursing, administrative and medical departments of the hospital.

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PHENOL MISTAKEN FOR ALCOHOL IN OPERATING ROOM

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ALTHOUGH a surgeon's lifetime experience is full of occasional accidental errors no one has ever witnessed such a misfortune in the operating room and therefore this case merits report.

M. T., female child, age five years, was admitted to a hospital with a diagnosis of acute appendicitis. The patient was operated on within the hour, and an acutely inflammed, pelvic appendix was removed. After the closure of the skin, the interne asked for an alcohol sponge to remove the excess iodine. Instead of the alcohol sponge, he was handed one soaked in pure phenol. Following its "vigorous" use, we immediately noticed cauterization and whitening of the skin over an area about 5 cm. in width surrounding the operative field (Fig. 1). Realizing the error, alcohol sponges were immediately applied.

For forty-eight hours, the skin was heavily coated with butesin picrate, and thereafter radolatum was used. Within twenty-four hours, the skin turned brownish-black and within seventy-two hours, blisters were noted. By the eighth day, portions of discolored superficial layers of the skin were removed, and surprisingly, fairly healthy skin appeared. Within two weeks after the accident all crusts were removed, only a pinkish hue remaining to the new skin which disappeared within the following week.

The postoperative condition otherwise was uneventful. The patient's hospital period was ten days.

The meager reports of such an accident occurring in an operating room, and the anxious days spent by the medical and nursing staff prompted me to report this case. The lesson to be learned is that when phenol is applied to the skin and immediately neutralized with alcohol, necrosis, ulceration and gangrene will not occur.

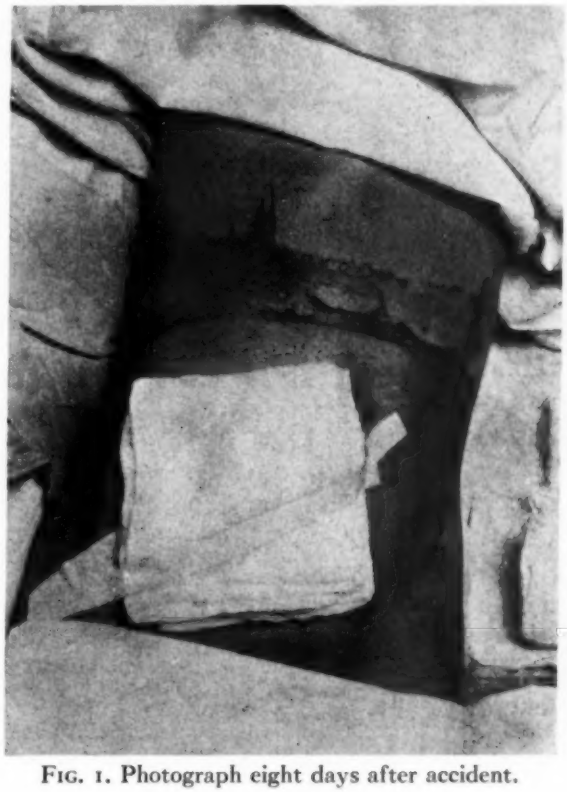

FIG. 1. Photograph eight days after accident.

NEW INSTRUMENTS

PERITONEOSCOPY

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VISUALIZATION of the peritoneal cavity and its contents through a scope was first accomplished in 1901 by Kelling, a surgeon of Dresden. He reported this "Zoelioskopie" to an assembly of scientists and physicians held in Hamburg. In 1910 Jacobaus, of Stockholm, applied the name laparoscopy to a similar method of clinical examination in man.

For various reasons this procedure has not met with the universal adoption it deserves, although it has been used successfully in various parts of the world and highly praised in many published articles in Germany, England, the Americas and other countries. Some of the reasons why the procedure has failed to become of general use are as follows:

1. No special perfected instrument has yet been placed on the market, although one has been devised by Doctor John Rudock of Los Angeles, California, and Mr. Wappler;

2. The difficulty of obtaining biopsy specimens; this can now be performed by using a cystoscopic rongeur forcep and the site from which the specimen is removed can be cauterized by a mild, diathermic spark;

3. The nonfamiliarity of the majority of the uses and possibilities of the modern cystoscopes.

In the numerous articles published the authors have suggested various types of instruments. However, these were all optical instruments, analogous to electrically lighted cystoscopes.

Immediately following Stiner's* excellent description of the procedure in 1924, I conducted experiments on 12 living dogs, using the Brown-Buerger right angled cystoscope and the flexible cannula and trocar as devised by Nadeau and Kampmyer of Chicago. I found it to be safe, simple, and to possess diagnostic possibilities unequaled by any other method except exploratory laparotomy. Unfortunately, many of the diagnostic methods used today in determining intra-abdominal and pelvic conditions, produce vague and only presumptive conclusions. Roentgenography has helped markedly, but peritoneoscopy offers further aid not obtainable short of exploratory laparotomy. It will not replace such laparotomies, but is the choice in many instances.

After employing this procedure with success on a few human cases I had a special gastrodiaphane made, differing from the Einhorn gastrodiaphane in that it has an air passageway which permits dilation of the stomach as well as transillumination. This, employed with the scope, permits study of the transilluminated anterior wall and greater curvature. It can also be used to balloon and transilluminate the sigmoid. This has proved of value in the diagnosis of gastric ulcer on the anterior surface near the lesser curvature and in gastric carcinoma and has helped to decide operability or nonoperability of cases. In one instance it showed a carcinoma in the anterior wall not discernible by gastroscopy or *x*-ray

* STINER, O. P. *Surg. Gynec. and Obst.* (April), 1924.

examination but which was the primary condition responsible for Krunkenberg tumors.

formed. This last method produces the same results as Dickinson's method, as devised in 1916. However, Dickinson's

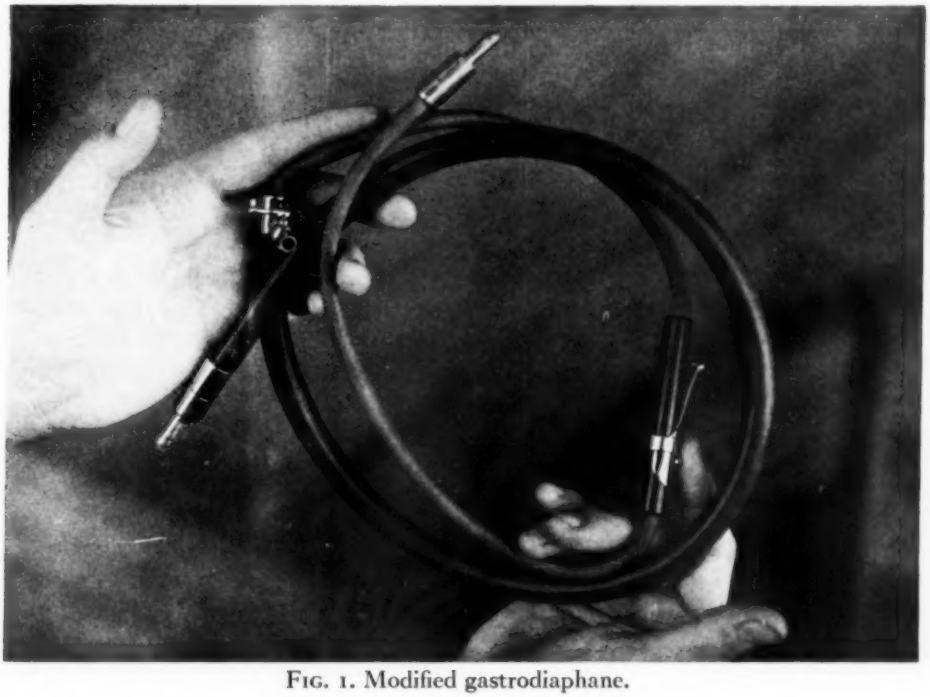

Fig. 1. Modified gastrodiaphane.

Following the appearance of the panendoscope I have employed it, using the 28 F. gauge sheath. Recently a special bistoury pointed obturator was made for me to fit my 28 F. scope. This is an improvement as it simplifies the work. By employing two instruments, one in the bladder and one in the peritoneal cavity, with the bladder gently ballooned with air, portions of the bladder wall may be studied by transillumination. Adhesion bands in the abdomen can be severed the same as bands of like character in the thorax. Simple cysts of the ovaries that produce symptoms from pressure on fibrotic tissues can be incised with a cutting current and knife electrode.

This work should be of special interest and value to the gynecologist as it allows ocular examination of intrapelvic reproductive organs and will help to decide the location and the variety of many masses found on bimanual palpation. Sterilization of the female, using my special electrode and endothermic coagulation can be performed. This last method produces the same results as Dickinson's method, as devised in 1916. However, Dickinson's instrument was passed transuterinely and guided by sense of touch, while my method is done under the guidance of sight and touch.

With increasing familiarity of the technique other minor procedures will likely be performed. Radon needles may easily be implanted in suspicious tissue, allowed to remain there the necessary treatment time and removed by traction on the silk threads attached thereto. It is now believed that peritoneoscopy will in the near future become a necessary part of modern abdominal and pelvic surgery. This is a safe and simple procedure with practically no discomfort to the patient unless pressure is exerted on unanesthetized peritoneum; pain sometimes results from unnecessary traction on the loops of the intestines.

TECHNIQUE

No preparation is necessary except that it is advisable to give morphine, grain ¼, twenty minutes before the puncture is made. The patient may have his usual

breakfast unless gastric transillumination is to be employed.

The procedure is to be carried out under asceptic operating room technique.

The patient, lying in the horizontal position, is prepared as for any laparotomy, draped, and all instruments thoroughly sterilized. The site of the puncture is selected and anesthised with novocaine. It is advisable to encircle the puncture site with subcutaneous wheals of novocaine solution with a diameter of about 10 cm.

Following this the needle is inserted to the peritoneum and infiltrated just above it. A half inch incision is made through the skin and anterior fascia. A dull pointed paracentesis needle is passed into the cavity, which is inflated with air by attaching to the needle a rubber tube with a bulb at its distal end. This causes the parietes to balloon away from the intestines. At first various gases were employed but later, filtered air; now we are using unfiltered air.

I have made it a point to enter through the body of one of the rectii muscles, believing that the natural contraction of muscle fibre is an added safeguard against possible hernia.

The sheath with the bistoury tipped obturator acting as a trocar is now passed into the cavity. The obturator is removed and the telescope and light inserted. Considerable amounts of the air first introduced will escape as the obturator is removed. Reinflation is performed through the irrigating channels of the scope. In case of ascites the inflation is omitted until after the fluid contents have been removed. Draining the fluid under guidance of the eye frequently enables us to avoid blockage of the tube by the omentum and also permits emptying of pockets.

With everything thus in position the light in the scope is turned on and the inspection begins. It is well at this point to select some one spot for orientation and then to systematically examine all possible points, reserving for the last the thorough study of apparent pathological foci.

An all position operating table is of marked value in this work. By inflating the peritoneal cavity the intestines fall away into the more dependent portions of the abdomen. By shifting the position of the patient the various fields are thus exposed. With the patient in the Trendelenburg position the female reproductive organs stand out in bold relief. The Fowler position allows visualization of the upper abdominal organs. The undersurface of the liver can be gently uplifted with the scope and the gall bladder inspected. Stone can not be palpated in all cases but I have been successful in so doing in 4 instances. The tip of the scope may be used to push loops of the intestines aside, thus improving the field of the view. Also the omentum can then be studied more thoroughly.

Upon removal of the scope, after the abdomen has been deflated, a suture is unnecessary. The muscle fibres will prevent any hernia. The skin incision is closed with sterile adhesive tape. In the work on dogs a reentry in three or four weeks time showed that the original wound had healed with almost indistinguishable scar. There will be a small amount of air retained in the abdominal cavity but this will do no harm for in a few days it will be spilt into its chemical elements and absorbed.

In case transillumination of the stomach is not desired, simple distention by means of a Rehfuss tube may assist in diagnosis. Likewise, transillumination of the rectum and sigmoid or simple distention in some cases may be of value.

The picture presented by transillumination of the vaginal vault is surprising. The work should be conducted in a dark room as the location of the tip of the instrument is thus easily checked by the light shining through the abdominal wall. A gynecological examination can be aided by inserting the hand into the vagina and manipulating the pelvic organs. Tumor masses may be lifted into view. With a hand on the abdomen surface manipulations may assist.

The appendix is seen only occasionally. With the patient in the Fowler position the liver falls away from the diaphragm and can be examined as to color, smoothness, nodules and size. Enlargements of the spleen may be easily noted. After a few examinations the picture becomes clear, natural and easily understood. Only the peritoneal surfaces are seen. Shadows, enlargements, discolorations and nodulations are brought out by transillumination.

This procedure should not be employed in acute conditions where the possibility of infection exists, but should be used in chronic conditions where doubt exists as to the probable etiological factor.

SUMMARY

1. Peritoneoscopy is a safe and simple procedure of marked value in abdominal and pelvic diagnosis, especially in chronic conditions.

2. Dilatation and transillumination of the stomach with the modified gastrodiaphane permits studies to be made that are not available by any other method.

3. It is my belief that these procedures will soon become a recognized standard technique.

A PRACTICAL COLOSTOMY IRRIGATING APPARATUS

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INDUBITABLY there are numerous phases in the management of colostomies. Many times well made ones cause very little trouble, yet again neither well nor poorly made colostomies function as precisely as one wishes and minute instructions must be given for their care. Too, there are patients who wish to attend to their colostomy toilets once a day and then not be hampered with further thoughts of their hygiene. The apparatus presented herein may be used in either instance. It is also a useful appliance for hospitals in cases where the patient may turn on his side or may assume a sitting or standing position. It has been my experience that it is occasionally very difficult to give colonic irrigations through a colostomy with an ordinary syringe and kidney basin, and it becomes a rather messy procedure for both the patient and the attendants.

This apparatus is made from what might be called the old type of urinal. A metal tube one-fourth inch in diameter runs from a point just behind the handle out through the center of the opening. A douche bag or irrigating can is attached to the end of the metal tube behind the handle. A short piece of rubber tubing or a catheter is attached to the opposite end of the metal tube for insertion into the colostomy opening. A circular plate fits snugly into the neck of the urinal and is constructed so as to slide back and forth on the metal tube. This is controlled by a small handle projecting on the outside and acts as a valve for controlling pressure and the amount of distention and height of irrigation. It can be easily manipulated

FIG. 1. A metal tube ¼ inch in diameter runs from a point just behind the handle down into the container and out through the center of the circular opening, extending about ¼ inch beyond the brim. To this is attached a small catheter or rubber tube which is to be inserted into the colostomy opening. A douche bag is attached to the metal tube behind the handle. A metal diaphragm, A, slides along the inner metal tube.

with the right hand. As the handle is pushed forward, the valve closes the neck of the container, forcing the fluid back into the colon. When the patient feels sufficient distention, the handle is pulled back and the contents flow back into the urinal.

This valve was not used in early experiments with this container and the apparatus proved inadequate at times because the colon was not stimulated enough to become active and thus completely empty itself. Under pressure, the solution so distends the colon that upon release of the valve it is natural for the colon to contract and expel the fluid. The contents of the can then may be emptied into the toilet and the container may be washed by removing the cap on the end.

In those cases where it seems necessary because of frequent discharge from the colon, or where an individual prefers to have a selective time for cleansing the colon in order to be free for eighteen or twenty-four hours, I usually advise performing the irrigation before breakfast. The colostomy is then protected by a dressing of cellucotton covered with a piece of waxed paper and a colostomy belt or binder such as has been described previously by the author is worn. By the time one has finished breakfast, any remaining fluid has usually been released from the colon. The dressing

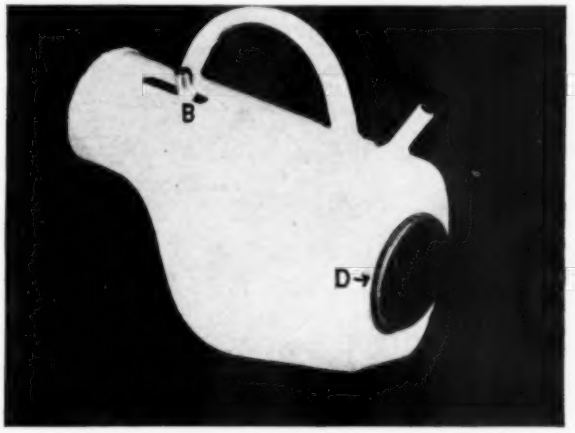

Fig. 2. The lever, B, controls the metal diaphragm which opens and closes, controlling the pressure and preventing backflow from the colostomy into the collecting can. By unscrewing the metal plane, D, the inside of the can may be thoroughly washed.

may then be changed and the patient can ordinarily go twenty-four hours with perfect confidence and with no further thought of colon discharge.

Colostomy patients, like patients with normal gastrointestinal tracts, are subject to attacks of diarrhea and in such instances this apparatus has proved invaluable.

[From Fernelius' Universa Medicina, Geneva, 1679.]

BOOKSHELF BROWSING

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SAN FRANCISCO

WORLD progress requires separating from set dogmas and theories, particularly from any fetish slavery to that knowledge which is stipulated in books. There exists in books or rather in the printed matter therein, a great point of weakness, which dates back four to five centuries to that epoch when the transition was being made between the handwritten manuscripts and the printed page. Many of these manuscripts were deliberately and necessarily well hidden by their owners because they contained knowledge banned by the religious or political authorities of the time. A necessary step in this transition was that of translation from the old Latin, Greek, Arabian or Egyptian into the printed form in a modern language. It was easily possible for error to have crept in at any step in this process. This was even more possible when it is considered that the bulk of the translations were done by monks or friars either cloistered in a monastery or journeying over the land as travelling mendicants. These monks or friars were human beings, subject to the same mental, physical and emotional stimuli, as other ordinary humans, and it is permissible to speculate whether a certain line or passage in the Greek would have exactly the meaning the original writer intended if the translator had had a sleepless night or was having a bilious attack or a severe headache at the particular time of translation. Then again a group of even more unreliable translators consisted of a great many of the lesser fry among the so-called wise men who were the mountebanks, the bluffers and fourflushers of their time. They made a great pretense of knowledge which they did not possess, but had to maintain this pose by an egotistic bravado accompanied by a tremendous barrage of words, the greater part meaningless. Imagine what one of these knaves might do in translating some ancient script into modern tongue to suit himself and at the same time cover up his intellectual deficiencies.

It is well worthwhile to have a mental visualization as to the exact modus operandi of the transition of a manuscript into the modern printed word. Perhaps over one-half of this work was done by the members of three great religious orders, the Benedictines, a more liberal group who encouraged translations of all works whether or not religious, the Cistercians and the Dominicans. The latter were a bit biased, encouraged only religious works or

those having some bearing on religious philosophy and usually suppressing everything that did not exactly coincide with their own religious teachings.

In the monasteries of these orders, and each was about the same, there would be a darkened room, sometimes subterranean, in which the collection of manuscripts was kept in charge of one monk alone who was charged with their safety and care. Then there were small cell-like bare rooms or studies in which the scribe or translator worked entirely alone and uninterrupted as silence was considered absolutely essential to such momentous pondering. Also all work was done by candlelight as it was believed that daylight injured the manuscripts. Some of these scribes worked heart and soul, fired with religious fervor, and turned out enormous amounts of work; others of course were weary or resentful or humorous. Human emotions then were the same as today and these emotions were of course reflected and translated into their work.

There also existed lay scribes, who were not members of monastic orders and were much less reliable. They were notoriously poor, usually had an axe to grind and their feelings and beliefs crept into their work at the slightest opportunity.

Because of these conditions, a number of personages arose who would not accept the printed word blindly. Being of enquiring types of mind, they chose to think for themselves and when these minds were above average in acumen they attracted attention, just as any brilliant mind will attract attention whether in the fifteenth or in the twentieth century. There were many of this type who chose to challenge the theories and dogmas of Aristotle, Galen, Copernicus, Albertus Magnus, Thomas Aquinas and others, particularly as handed down in these translations, and among these was Cornelius Agrippa Von Nettesheim.

It is most unfortunate that the contributions of a great many brilliant men and minds of the fifteenth and sixteenth centuries were lost to subsequent generations because of the almost continuous political and religious strife present in

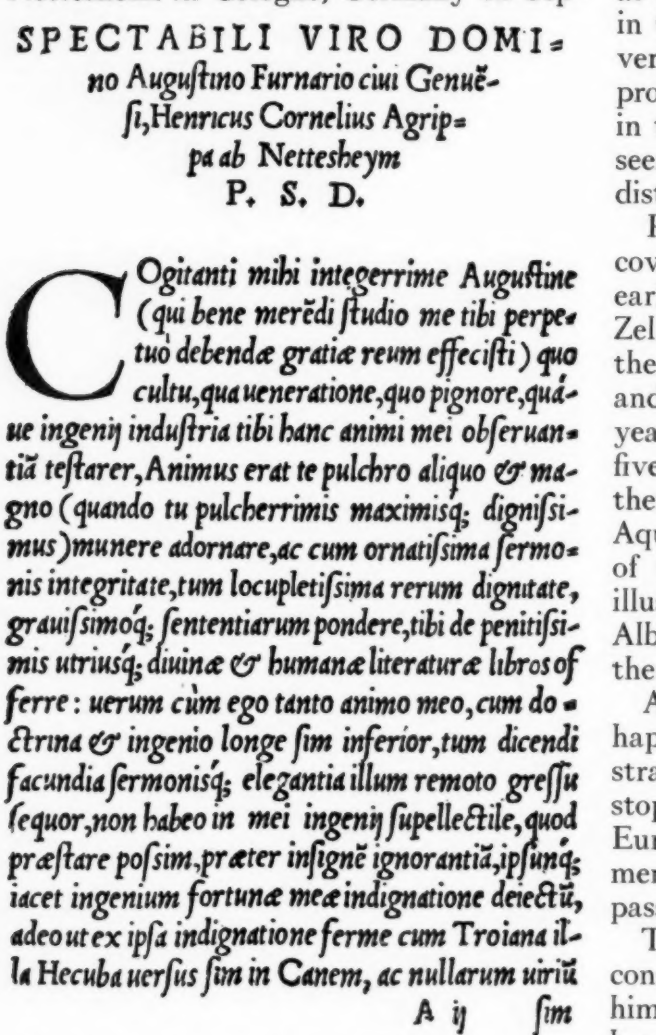
Fig. 1. Portrait of Cornelius Agrippa from a first edition of his "Vanity of Arts and Sciences."

Europe at that time. If a man wrote something which offended the state, he was cast in prison, his writings proscribed, and all his manuscripts or books buried, even though these might treat of scientific rather than political matters. If he wrote anything which offended the church, he was denounced as a heretic, burned at the stake and all his writings burned with him. This was the fate of Servetus and his writings with regard to the circulation of the blood, which probably antedated Harvey by a considerable time.

It was in such a manner that the true scientific worth of Agrippa Von Nettesheim was lost in the literature of subsequent generations and was minimized because of the amount of contempt and ridicule heaped upon it through superstition and crass ignorance.

However, he was not completely lost to sight, since Rabelais in his works speaks of him, Southey wrote a ballad concerning an incident in his life and Butler devoted considerable of his writing endeavoring to ridicule him.

But a bit as to the man himself. He was born a noble, of the royal house of Nettesheim in Cologne, Germany on Sep-



SPECTABILI VIRO DOMI=
no Auguſtino Furnario ciui Genuẽ=
ſi, Henricus Cornelius Agrip=
pa ab Nettesheym
P. S. D.

COgitanti mihi integerrime Auguſtine (qui bene merẽdi ſtudio me tibi perpetuò debendæ gratiæ reum effeciſti) quo cultu, qua ueneratione, quo pignore, quáue ingenij induſtria tibi hanc animi mei obſeruantiã teſtarer, Animus erat te pulchro aliquo & magno (quando tu pulcherrimis maximisq; digniſsimus) munere adornare, ac cum ornatiſsima ſermonis integritate, tum locupletiſsima rerum dignitate, grauiſsimóq; ſententiarum pondere, tibi de penitiſsimis utriusq; diuinæ & humanæ literaturæ libros offerre: uerum cùm ego tanto animo meo, cum doctrina & ingenio longe ſim inferior, tum dicendi facundia ſermoniſq; elegantia illum remoto greſſu ſequor, non habeo in mei ingenij ſupellectile, quod præſtare poſsim, præter inſignẽ ignorantiã, ipſunq; iacet ingenium fortunæ meæ indignatione deiectũ, adeo ut ex ipſa indignatione ferme cum Troiana illa Hecuba uerſus ſim in Canem, ac nullarum uiriũ

A ij ſim

Fig. 2. Dedication page from "Occult Philosophy."

tember 14, 1486. Apparently life was destined to be interesting from his very birth. Biographers have been unable to account for the Agrippa in his name, but Morley, his foremost biographer stated that as late as 1700, the term, "agrippa," derived from the Roman, was used to designate one who had been born feet first and he assumed that the name Agrippa had been applied to him because of this fact.

In his very early years he demonstrated a rare power of memory, an extreme aptitude for study and particularly a flair for languages, and he later became thoroughly versed in all European languages. Added to these assets were his position as the son of a noble family, his residence in Cologne, which at the time was at the very height of its commercial and scholastic prosperity, with a university well regarded in the centers of education, and it can be seen that the stage was well set for a distinguished career.

Printing had only recently been discovered and Cologne possessed one of the early presses and a typographer, Ulrich Zell, who ranked among the foremost of the time. He had started his press in 1463 and during the course of the next forty years had produced in printed form over five hundred different treatises. Among these were many of the writings of Thomas Aquinas and of Albertus Magnus, both of whom had spent some part of their illustrious lives in Cologne, particularly Albertus Magnus, who had been born there.

Agrippa's flair for languages was perhaps aided by the fact that Cologne was strategically situated as a crossroad or stop-over on the main great highway of Europe so that merchants and business men of many countries and many tongues passed through here.

This knowledge of languages also had considerable influence on his life, causing him to devote many years that might have been more profitably spent in study, to political and state matters, for which he was obviously unfitted as subsequent experience demonstrated.

As soon as he had finished his university studies it came to the attention of the Emperor Maximilian I, that Agrippa was so well versed in languages that he would be most valuable at court, so he was immediately drafted as secretary to the Emperor.

As the chief occupation of Maximilian was that of conducting wars over all of Europe, he could make good use of a secretary fluent in languages as a courier or messenger. Being himself quite proficient in languages he used Agrippa more

as a sort of secret service man, spy courier and confidential agent.

For a number of years, then, we find him engaged in political revolts and uprisings, some successful in outcome, others disastrous, following a life for which he did not care and for which he was not fitted.

During the course of some of these military adventures however, there came some compensations to his intellectual life. One of the greatest Greek and Hebrew scholars of the time was Reuchlin, and during the course of one of his military missions Agrippa met this scholar and remained for some time in his home studying the original Latin classics, the original Aristotle in Greek and the Bible in Hebrew.

On another of these military missions, this time to Lyons, in France, he encountered one of his former fellow conspirators in some political enterprise who introduced him at the court of Margaret of Ghent. Here the court were so astounded at his knowledge of Greek, Hebrew and Latin that he was offered a position at court, an appointment to the University faculty and the degree of Doctor of Divinity.

This suited him because he wished to abandon his life of travel and danger and settle down to serious study, and secondly, he wished to marry, so he accepted the appointment and almost immediately married a Jane Louisa Tyssie of Geneva, who was considered sufficiently intellectual to uphold her own and in the high and lofty intellectual life of her husband and was

THIS TOMB

SCARCELY THE GRACES KEEP, BUT THE BLACK DAUGHTERS OF HELL;
NOT THE MUSES, BUT THE FURIES WITH SNAKES SPREAD ABROAD.

Alecto

COLLECTS THE ASHES, MIXES THEM WITH ACONITE,
AND GIVES THE WELCOME OFFERING TO BE DEVOURED BY

The Stygian Dog,

WHO NOW CRUELLY PURSUES THROUGH THE PATHS OF ORCUS,
AND SNATCHES AT
THAT OF WHICH WHEN ALIVE HE WAS THE COMPANION,
AND HE LEAPS UP AT HIM.

And He

SALUTES THE FURIES BECAUSE HE HAD KNOWN THEM ALL,
AND HE ADDRESSES EACH BY HER OWN NAME.

O WRETCHED ARTS,
WHICH AFFORD ONLY THIS CONVENIENCE
THAT AS A KNOWN GUEST HE CAN APPROACH

THE STYGIAN WATERS.

So like a Pagan spat the Monk upon the Christian's grave!

FIG. 3. Translation from the Latin inscription upon his tombstone erected by the Monks.

considered a great beauty as well, a most satisfactory combination.

Having adopted the studious life, within a year he published his first treatise, on Magic. This consisted mainly of translations from the original of Albertus Magnus, contained much of alchemy, considerable medicine and some reference to the use of metals in the healing of the sick, particularly gold, mercury and antimony.

A few of the medical aphorisms published in this treatise follow.

1. Many things are possessed of the property of enmity, in which it angrily shuns its contrary, or drives it away from its presence. Such is rhubarb in its action on bile, treacle against poisons, powdered amethyst against habitual drunkenness.

2. In the colic, if a live duck be applied to the stomach it takes away the pain and the duck dies.

3. In the use of parts of animals they should be taken from the animal while it still lives and is in its fullest vigor. (A good endocrine observation, but hardly of any modern use, what with antivivisection societies, etc.)

4. It is well known among physicians that the eating of brains helps the brain and the eating of lung helps the lung.

Apparently the life of study and letters was that most suited to him as we find in another year the publication of another treatise, this time on occult philosophy. This contained observations of his own but in the main contained translations from the old Arabian physician, Averroes. A few quotations from this work follow:

Physicians must own a belief in faith and that faith is more potent for cure than even medicine and by having a strong belief in their own power of curing, they give new and added strength to their remedies. Distrust and doubt will break the power of a physician's mind and frustrate him in bringing about the desired result.

That passage is worthy of inclusion in any baccalaureate sermon.

Another of his own observations:

The effect of melancholia upon men is peculiar; when the mind is forced beyond the bounds of reason it is possible for a totally ignorant person to become an artist; if he is a prophet, he may prophecy great disturbances in the elements, and of the work of men in ages to come, he may even become a famous philosopher, or a famous orator.

One of his translations from Averroes:

The senses of men are divided into four:

1. Common sense, that one which collects and perfects the impressions brought in from without.

2. Imaginations, that which takes and retains impressions and presents them to,

3. Fancy, that which judges what the things are, of which representations are thus brought to it, forms opinions upon them and gives them to

4. Memory, that which keeps all these.

By dint, therefore, of his writings and his fame as a scholar versed in the ancient as well as the modern languages, he came to the attention of the University of Pavia, which, in 1514, when he was thirty years of age, honored him with the degree of Doctor of Medicine.

The actual practice of medicine did not seriously at any time enter his mind. He preferred to remain purely a scholar, delving deeply into philosophy with which the medicine of the time was so firmly united, translating and bringing to light old manuscripts, correcting modern translations, etc. It is in this field that he rendered service to medicine.

For a short time he did attempt to practice for monetary profit but was dismally unsuccessful. He also became rather riskily involved in the great Lutheran movement gathering headway in Europe at the time and wrote many treatises in support of Luther. At times this activity came near causing the loss of his life.

In 1522, eight years after receiving his doctorate in medicine, he was described in a letter by Reuchlin thus: "a man more learned than any, by profession a physician, but of all knowledge at the same time a

cyclopedia, strong in disputation, a native of Cologne, an Italian by education."

Economic necessity and the death of his wife forced him to leave Geneva and accept the post of physician to the town of Fribourg, not a remunerative post as the town was small and unable to pay a high salary, but as he wrote later, made up for lack in salary with the generous esteem and admiration in which it held him.

He was next offered the post as physician to the Queen Mother of France, which he accepted with some misgiving, as the question of remuneration was extremely hazardous and although the honor was great, he could not live and eat on honor alone. But he left Switzerland for France and was given quarters in Lyons.

It was while at Lyons that he wrote his "Vanity of Sciences and Arts," of which the title page of the original edition is reproduced (Fig. 2). That the spirit of the book is satire can be judged by the motto: "Nihil Scire Felicissima Vita" (Ignorance is Bliss). It is in this book that he challenged the custom of the great universities to swear their disciples never to contradict Aristotle, Boethius, Aquinas and Magnus. He wrote,

> You tie down the road to free inquiry, you chain venturesome spirits to the ground, you claim to have all wisdom, you claim to be the fountain heads of wisdom, but you are not so wise as you account yourselves, for I can find more in praise of an ass than any one of you. If any scholar differs so much as a nail's breadth from your teachings, you claim him a heretic, a criminal against holy science and doom him to fire and flames. What a piece of tyranny to deprive a scholar of the liberty of searching for and following truth.

In the chapter devoted to "Physic" he wrote that it was another art of homicide though claiming the name of a philosophy. He cited the different factions into which physicians were divided and the bitter controversies between each.

Then he paraded the uncertainty of their diagnoses and cited many notoriously erroneous ones. The pomps and vanities of physicians, the manner in which practice accrued to him who owned a velvet coat or rings of gold or who either by a certain display of religion, or by unparalleled self assertion used Latin sentences and names of which he did not know the meaning, these were the subject of satire in this book. He satirized many of the little tricks of the day and investigation shows that they are about the same tricks as of today. He ridiculed the tickling of the palate of the effeminate and feminine by prescribing dainty tidbits, maintaining that this was not the practice of medicine, merely a by-way to acquiring a practice. With scathing remarks he depicts the pompous attitude of the doctor toward the pharmacy, and the affectation of the doctor in ordering that the medicine shall be compounded before his own eyes, as if on him and him alone rested the correct compounding, pretending at times to personally select the ingredients when as Agrippa states, for the most part, he knew not good from bad, in fact, hardly knows or recognizes the herbs and chemicals themselves when he sees them.

He goes on to describe the great commotion over commending that the patient must have unusual things and the to-do about prohibiting common things, such as the use of far-fetched and costly medications and those in which the medical profession has fostered the belief that they can only be effective according to the time of day or night when they were gathered, adding, who knows at what time of the day they were gathered by unscrupulous venders, totally unreliable, and how ridiculous of the profession to foster such an idea.

He then volunteers a bit of advice. Why not use the simples of the country, which God caused to grow there as the proper antidotes to the diseases of that country, and he cites that there have been famous philosophers who have not considered it beneath their dignity to write

treatises upon these simples, such as Chrysippus wrote upon colewart, Pythagoras upon squill, Marcion on the radish, Diocles on the turnip, Phanius on nettles.

I wonder if any of these old master minds wrote about carrot or spinach or if it remained for the present generation to first learn of their efficacy under the imposing heading of vitamins?

He pleads that there should be a law similar to that among the Egyptians of old, that in the first three days the physician was to cure a disease at the hazard of the patient, but after three days at the peril of his own.

Then he attacks the apothecaries because of their secret adulteration of drugs in order to make more profit. In a chapter on diet he stresses the contrary opinions of physicians as to diet, some stating that a certain food was injurious to the eyes, that cheese offended the stomach, milk offended the head, and the drinking of much water the lungs, and he ventured to say that in all the rivers, fields, gardens and markets, there could scarcely be found anything fitting for a man to eat, were one to follow these divergent views.

As can be seen these shafts of satire, although published at this time only in manuscript form certainly did not bring him a rush of popularity.

He therefore decided to quit France and after enlisting the aid of various friends he was offered the post of physician to the Queen Regent of the Netherlands.

The book "Vanity of Arts and Sciences" was then published in Antwerp and he was quite pleased to own a printed copy which he could pass around and loan his friends. He was able also to correct many printer's errors and to add more material to it.

An example of added material:

> Many men seek a knowledge of the arts and sciences mainly in the hope that a defect of natural parts may be supplied by the help of acquired ones. As if it were probable that Nature, which has been so exact and curious in the mechanism of flowers, herbs, flies, should have bungled most in her masterpiece and made men as it were by halves, to be afterwards polished and refined by his own industry.

His manuscript, "Occult Philosophy," was next printed in 1531 by John Grapheus in Antwerp and reprinted in Paris in 1532.

However, the "Vanity of Arts and Sciences" was causing him no end of trouble and making him many enemies, particularly among physicians, lawyers and priests, so that when his patroness died in 1532, they began to get in their work by denouncing him as a magician, sorcerer and addicted to witchcraft.

His income as physician to the Court was withheld and he found himself in debt. He hesitated to detach himself from the Court and maintain himself by means of his writings because it was risky to incur the displeasure of the reigning monarch. He found himself seized for debt and thrown into jail and released only through intercession of friends, and demonstrating that the only reason he was unable to pay his debts was because the royal treasury was holding back money due to him from the Emperor.

At the same time the Dominicans launched their attack against him, his books were proscribed and ordered to be burned. He was kept constantly defending himself from attack and the struggle was truly herculean because the attacks arose on many sides and on many indictments.

He wished to return to Cologne but so much had been stirred up against him and particularly against his book "Vanity of Arts and Sciences" that the Emperor ordered him exiled and he was not to return to Germany.

He lived about four months longer as a wanderer in France, dying in Grenoble on the way to Lyons, a hunted, persecuted and almost utterly forsaken man, at the age of forty-nine years.

Someone must have been entrusted with his manuscripts and his writings because

they were published in Lyons after his death with much of the material changed in order to meet with the approval of the priests.

Even in death, the revenge of the priests was insatiable. They built up a fantastic story to back their claim that he was a sorcerer and had erected upon his grave a marble slab with a Latin inscription, the best English translation of which is as follows:

His downfall occurred directly as the result of his critical treatise, satirizing the so-called learning professions, his "Vanity of Arts and Science." As we read his criticisms we may very honestly say to ourselves that these same foibles continue in our profession today, practically unchanged from his time. Would one be inclined after reading the trials and tribulations suffered by him because of his exposures, to accept the issues of today as a challenge, or to merely allow that such things exist and meekly abide. Much could be written about the science and pseudoscience that surrounds many of our own present day drugs and medicaments.

PRACTICAL CLINICAL GYNECOLOGY

BY

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PRACTICAL CLINICAL GYNECOLOGY

Chapter I

EXAMINATION OF THE PATIENT

BEFORE examining the patient a careful history should be taken. For purposes of accuracy and completeness a definite outline should be followed consistently, which soon becomes pure habit.

A careful, detailed history serves two purposes: (1) the immediate one of aiding in the making of the diagnosis; and (2) a remote purpose of furnishing data for future follow-up analysis and statistical study. The maintenance of complete, accurate histories and follow-up notes is of great help in the proper evaluation of one's work and end results.

It is understood that a general physical examination, including heart, lungs and gross neurological, must be done on every patient. This is very essential. To operate on a patient for fibroids who has an active pulmonary tuberculosis or carcinoma of the breast would be poor gynecology.

No examination should be made on any patient in bed unless she is too ill to be moved. All examinations should be made in an examining room under good light, with good exposure and on a table that allows the body to be slightly flexed on the pelvis. Examinations made in bed with poor light and poor exposure lead to a great many errors of observation. *Most errors are made not from lack of knowledge but from lack of observation and improper interpretation of the findings.*

In making an examination gentleness cannot be overemphasized. The patient should be caused no pain or as little as possible; otherwise she cannot be examined properly and occasionally a too rough examination may cause injury or damage. *Be very, very gentle.* A little judicious "kidding" will go a long way toward helping the examination. Ward and

dispensary patients like all others, enjoy any extra consideration that is shown to them. One may acquire more information by gentle persistence than can be obtained with forcible, rough manipulation. When attempting to make a bimanual examination, talking to the patient and exerting a little gentle pressure on the abdomen will obtain more information than an attempt to overcome, by force, the resistance of the abdominal wall. Elevating the head and shoulders by tilting the table also helps relax the abdominal wall.

Never examine the immediate area of disease first. If the patient's disease is in the vulva, first rule out everything in the abdomen.

Every examination, like the history, must be made systematically by regular steps. If each patient is examined the same way every time, it will be but a short time when that routine examination will become second nature. The more closely one follows the same regular routine in every examination, the more rapidly will one's senses become trained and the fewer will be the errors of omission.

When the patient's chief complaint is pain in a definite localized area with a definite constancy or periodicity, it is advisable to see if one can reproduce the same pain in the exact spot. This may give a very definite lead as to the pathology that may be present. If, however, the attacks of pain are uncertain and indefinite, one should still try and examine the patient during an attack of pain. This may require a wait of twelve to twenty-four hours. For this purpose the patient should be hospitalized and the house surgeon instructed to watch the patient carefully so that when she has the particular pain he may examine her, and so elicit a possible lead as to the cause of the pain.

Always have the bladder empty by voiding before any vaginal examination. If a central cystic mass apparently in front of the uterus is palpated even after voiding, have the patient catheterized. *Don't be fooled by a distended bladder.* A distended bladder will cause more difficulty in diagnosis and operating than any actual abdominal pathology.

Inspection of the Patient

On first inspection, give a description of the general appearance of the patient. Does she look acutely or chronically ill? Is she pale, anemic? Is there anything specific about her general appearance? General inspection of the patient occasionally will make the diagnosis. Does she lie quietly, as in general peritonitis, or is she climbing all over the bed as in colic (renal) or twisted ovarian cyst? Observe the general appearance of the patient very carefully before making the local examination. *Train your eyes.*

Inspection of the Abdomen

Observe:

1. The size,
2. Skin markings,
3. Presence or absence of dilated veins,
4. Old scars from previous operations,
5. Old scars or new scars from pregnancy (the same sort of scars are frequently caused by adiposity).

The commonest swelling in the abdomen of a woman in the child-bearing age is a pregnant uterus.

Beware of the error of mistaking a distended bladder for an abdominal tumor, in the midline low down.

The condition of the umbilicus is fairly important in abdominal diagnosis. In a fat abdomen, the umbilicus is usually depressed and deep. If the size of the abdomen is due to an abdominal tumor, ascites or a large pregnant uterus, the umbilicus flattens out and loses its depression. Sometimes, one may see, particularly in patients with an umbilical hernia, blueness around the umbilicus in ectopic pregnancies. This is called Cullen's sign.

Considerable information can be obtained by watching the excursions of the abdomen on respiration. A patient with an acute inflammatory condition is not breathing with the whole abdomen. If the respiration is of the thoracic type and the abdomen does not move very much, one may suspect the

patient of having a ruptured viscus, ruptured pus tube, ruptured ectopic or generalized peritonitis. If the respirations seem thoracic in type and the patient is breathing by dilating

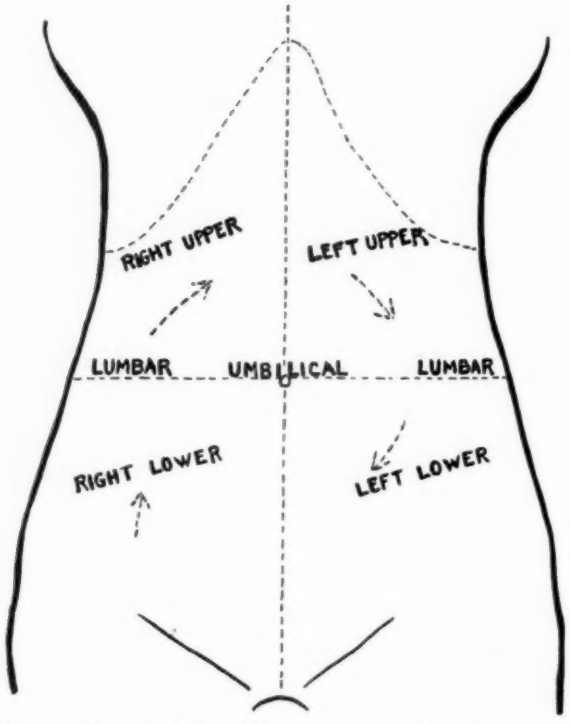


FIG. 1. Zones of the abdomen, arrow shows route to be followed.

her alae nasi, a pulmonary lesion should be suspected. Careful observation of such points as these and their proper interpretation help greatly in making correct diagnoses.

Often a tumor that is not palpated can be seen on respiration when the patient lies down and the abdomen becomes flattened. There may be individual areas of distention, particularly in patients who have had previous laparotomies, due to adhesions causing partial intestinal obstruction with localized distended loops and occasionally visable peristalsis.

Look for all these signs everytime and in each case. This sounds like a lot, but actually they can all be done in half a minute, if done regularly and systematically.

Palpation of the Abdomen

When palpating the abdomen do not palpate with a cold, rough, hard hand. A cold hand put on the abdomen gives "goose pimples" and causes the abdominal musculature to become tense. On coming in from the cold, the hands should be washed in hot water before being applied to the abdomen. Go over the entire abdomen gently, regularly and systematically, so as to find any special areas of resistance. Don't use the points of the fingers to make pressure, use the whole flat of the hand. One can obtain more information that way. Using the points of the fingers causes a tightening of the muscles. The patient should breathe lightly with her mouth open.

On palpating the abdomen *palpate the area of suspected pathology last.* Feel the patient in the right lower quadrant, right upper quadrant, region of liver, gall bladder, epigastrium, left upper quadrant, left lower quadrant, right and left lumbar regions, the suprapubic area, and the costovertebral angles. (Fig. 1.) Do it routinely so that no areas will be missed. A six months size pregnant uterus has been missed entirely because the examination was not made in this fashion, the entire abdomen was not gone over. The baby's head was felt and described as the uterus. In describing scars, masses, areas of tenderness, etc., divide the abdomen into zones. This helps materially in giving a more accurate description. All areas of tenderness, rigidities and masses should be mapped out on the abdominal wall in ink and then described. Mercurochrome in alcohol and acetone (Scott's solution) is a very good colored agent to use, it stays on the abdomen for some time and one is thus able to detect changes in the size of tumor masses. The fibroid charts have a diagram of the abdomen which can be used to map out the daily or weekly changes of the above lesions. In inflammatory lesions one is frequently able to study the changes in the size, shape, consistency, areas of tenderness, etc., and by the use of these outlines make daily records on the chart.

The description of an abdominal tumor should include its

General location	Mobility	Tenderness
Size	Consistency	Shape
Origin	Attachments	

Differentiation of Ascites from Ovarian Cysts

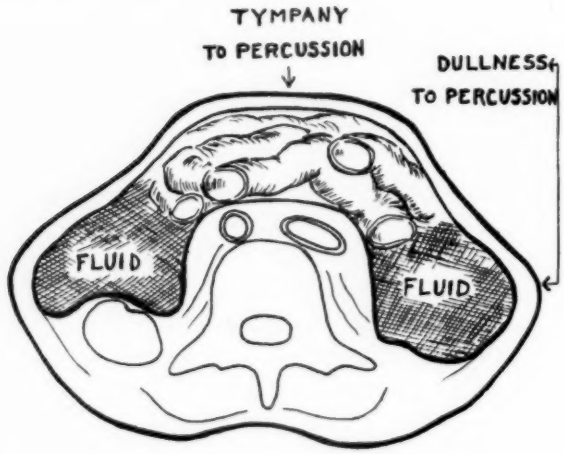


Fig. 2. Cross section of an abdomen with ascites showing the cause of physical signs.

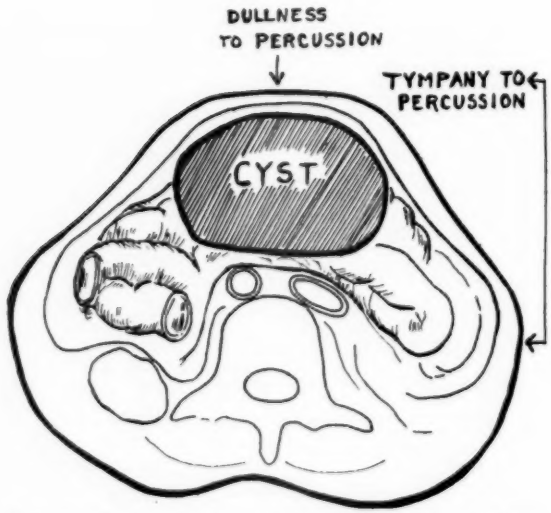


Fig. 3. Cross section of an abdomen containing a large cyst, showing the cause of the physical signs.

Ascites	Ovarian Cyst
1. Swollen abdomen	1. Swollen abdomen
2. Percussion resonance persists around umbilicus	2. Umbilicus usually dull to percussion
3. Flanks are dull to percussion	3. Percussion resonance occurs in the flanks
4. There is a fluid wave	4. Large, not tense ovarian cysts can give you a fluid wave; otherwise fluid wave is absent
5. When patient lies flat in bed there is usually billowing out of lateral portions of her abdomen	5. When patient lies flat you usually find a bulge in midline or somewhere in the anterior abdominal wall
6. Shifting dullness	6. No shifting dullness

Pelvic tumors, as a rule, cannot be displaced into the general peritoneal cavity and the examining fingers cannot be introduced below the tumor mass in the suprapubic or hypogastric regions. There are some exceptions; ovarian tumors or fibroids with long pedicles or a uterus held out of the pelvis by adhesions from a previous operation. A patient who has had a cesarean section may have had her uterus sutured high on the anterior abdominal wall. If that patient developed a fibroid, the examining hand could be introduced between it and the symphysis. One can usually follow these tumors up from the pelvis.

Vaginal Examination

The gynecologist must have eyes at the ends of his fingers. In order to do this he must make thousands of vaginal examinations and verify his findings by operation.

"How can I do it?" is a frequent question.

Never miss an opportunity to do a vaginal examination on a patient under narcosis. Make the vaginal examination in the anesthesia room and describe exactly, in writing, just what is felt. Stay for the operation and see what was felt and described and put both findings in writing; it helps fix the facts in the mind.

The question often arises, "How can one tell on vaginal examination the difference between a pus tube and an ovarian cyst?" Some say it cannot be done. Actually it can; if one examines patients very carefully under narcosis it is only a

matter of time before one will know the "feel" of an ovarian cyst or abscess. A cyst is usually globular, fairly sharply demarcated; whereas an abscess, although cystic in feel, if not tense, is not sharply demarcated from the surrounding tissue owing to the inflammatory exudate present. It is very important to write in a little notebook exactly what is felt and follow this by an accurate description of what was found at the operation. The fingers will in this way, develop a sense of differentiation that will be invaluable.

General Points about Vaginal Examinations. In making a vaginal examination the abdominal hand pushes the structures down to the fingers in the vagina. Everything should be felt with the vaginal hand. *Don't push structures from the pelvis up to the abdominal wall, but let the fingers in the vagina feel what is pushed down to them.*

When the two hands are fixed, their relative positions should not be changed or a false impression will be created.

When making a vaginal examination in women with long vaginas or if the examiners fingers are particularly short, it may be necessary to press very hard in order to have the fingers reach the end of the vagina, so that the various structures can be pushed down to them from above; this causes the transmission of a large number of motor stimuli along the nerves of the hand and forearm which reduces the sensitivity of the fingers. To avoid this, one of two things may be done:

1. Place the fingers in the vagina; have someone else push the elbow. In that way the pushing of the elbow and all motor stimuli will be done by someone else and the examiner's fingers will be absolutely passive (Fig. 4). Thus there will be no straining of the muscles and no dulling of sensations.

2. If the vaginal examination is made on an examining table the elbow can be pushed either by the knee or the hip, (Figs. 5 and 6) and in that way the motor stimulii down the arm are reduced to a minimum.

A menstruating woman need not be examined vaginally, but it is no contraindication. In her case, examination may be put off. *But a bleeding woman must always be examined.* Bleed-

ing is one of the earliest signs of severe pelvic pathology, sometimes very serious as carcinoma of the cervix or of the body of the uterus, and always requires investigation.

FIG. 4. Examiner's elbow being pushed by the hand of an assistant.

FIG. 5. Examiner's elbow being pushed by his knee.

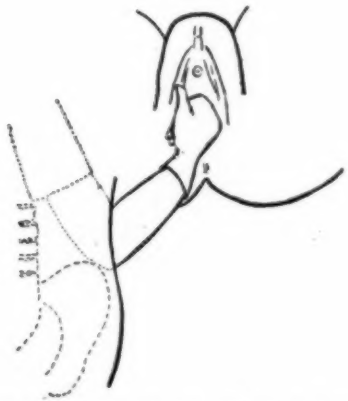


FIG. 6. Examiner's elbow being pushed by his hip.

Do not give a patient a douche or wash the external genitalia before making the first vaginal examination because this will frequently wash away valuable information. Feces or other discharges may be in the vagina or on the vulva, and thus aid materially in the diagnosis. If washed away an important diagnostic aid will have been lost. After the inspection has been made and smears, etc., have been taken, a douche can be given.

The bladder and rectum should always be empty, for when full they cause frequent mistakes in diagnosis. A word about differentiating a pelvic mass from feces: If on placing the

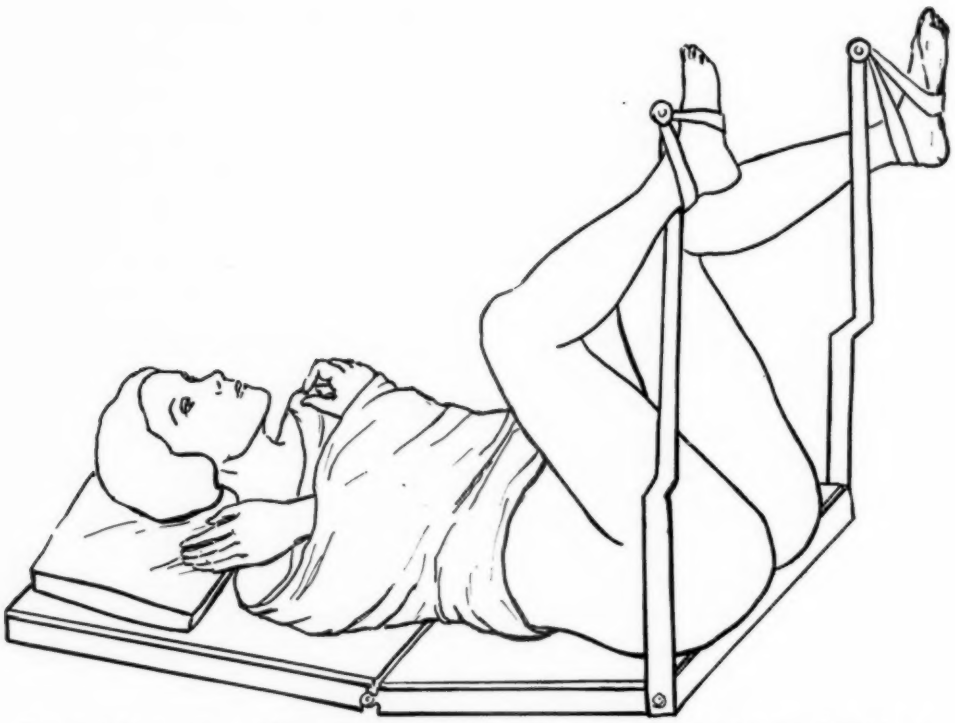


FIG. 7. Lithotomy position, buttocks to edge of the table, legs resting comfortably in stirrups; head and chest slightly elevated.

fingers into the vagina a pelvic mass is felt, push the finger into it, if the mass dents, it is fecal. If this does not establish the diagnosis, give an enema or a cathartic. *A dose of castor oil has cured many pelvic tumors.*

POSITION OF THE PATIENT FOR VAGINAL EXAMINATION. Lithotomy position is good for practically all vaginal examinations. The buttocks should be down to the edge of the table. Don't try to examine a patient whose buttocks are 6 to 10 inches away from the end of the table. Have the legs in some form of stirrup in which they rest easily, so that they are not under tension, and elevate the head and chest slightly so as to relieve tension on the abdominal wall (Fig. 7).

If a patient is to be examined for a fistula, some form of cervical lesions and particularly for vesical lesions, the knee-chest or the Sims' position is best.

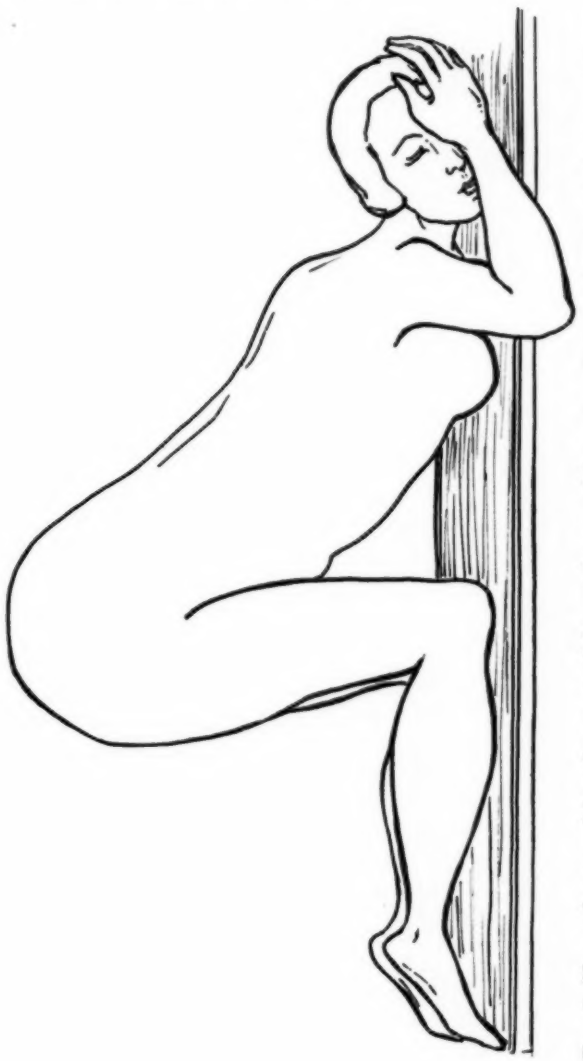


FIG. 8. Knee-chest position; knees at right angles to table and well apart, head and chest resting on the table.

In the knee-chest (Fig. 8) and the Sims' position (Fig. 9) on separating the labia, air rushes into the vagina and the vagina balloons out; thus all its walls can be examined with a Sims' speculum very readily. In the lateral Sims' position the buttocks should be elevated so that there is a curve in the back. In the knee-chest position the thigh should be at right angles to the table and the knees should be well apart.

INSPECTION OF THE VULVA AND LABIA. Observe alteration in form, color of the vulva, scratch marks, swellings, size and thickness of the labia and any ulcerations that are present.

FIG. 9. Sims' position; buttocks elevated so as to have a curve in the back, right leg flexed as far as possible, left knee slightly flexed.

If ulcerations or any palpable masses are present always examine the inguinal region for glands. When the labia are separated describe the type of discharge that is present. The urethral orifice should be observed to see if it is everted, red, sensitive, if there is any discharge present; if there is, take a smear. Milk the meatus to see if there is a discharge present. Feel for the Bartholin's glands and if enlarged, describe them.

THE VAGINAL EXAMINATION. For the ordinary vaginal examination use the lithotomy position; introduce two fingers, well lubricated, into the vagina. *If a smear for trichomoniasis*

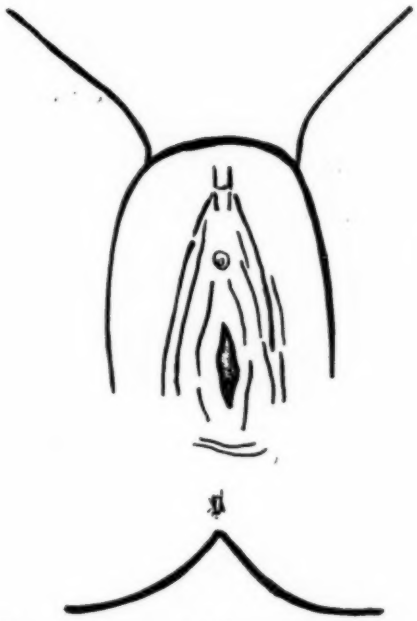


FIG. 10. Entrance of the vagina is seen as a vertical opening.

FIG. 11. Cross section of the vagina showing its axis to be transverse, roughly "H" shaped.

is to be taken, use no lubricant on the fingers as any kind of lubricant destroys trichomonas. In all other cases the fingers should always be well lubricated before being placed in the vagina. The best lubricant is diluted tincture of green soap; it does not stain, leaves no marks, is easily removed, is clean, is easy to obtain and organisms do not grow in it.

When passing the fingers into the vagina, do it without pain to the patient. Most examiners forget that the entrance to the vagina is vertical and that its inside axis is transverse (Figs. 10 and 11). The fingers must be put in at an angle by depressing the pelvic floor. All pressure must be exerted downward so that nothing presses against the anterior vaginal wall, urethra and symphysis. The vestibule is very sensitive.

Once the fingers are in, they are rotated and introduced in the transverse diameter of the vagina.

On first placing the fingers in the vagina, test and describe the condition of the pelvic floor. A lacerated perineum does not necessarily mean a rectocele, for a complete laceration of the perineum may be present without a rectocele.

Separate the labia well and with the two fingers in the vagina, exert pressure downward on the pelvic floor and have the patient bear down. If there is a cystocele present the bulge is above the fingers, a rectocele will be below them.

In examining the vaginal walls describe the rugae, any pathological areas or roughness, warts, cysts, ulcerations; also note the presence and depth of the vault of the vagina. This is important for two reasons: It is not at all unusual to find in sterility cases that the vagina comes directly downward from the cervix and there is no receptaculum for the semen. In congenital retroflexions one frequently finds a short anterior vaginal wall with very little anterior cul-de-sac, thus the cervix is drawn forward and as a result the fundus of the uterus is thrown back, a fact to remember, particularly in vaginal repairs of the anterior wall where, if the wall is markedly shortened from before backward, it may cause a retroflexion, which may give more trouble than the previous cystocele.

Following the examination of the vagina one feels for the uterus placing the other hand on abdomen. If the cervix is tapped slightly, this will aid the abdominal hand in locating the fundus. With the middle finger behind the cervix and the opposite hand on the fundus, thereby fixing it, the index finger can be slipped in front of the cervix and the anterior uterine wall, thus the whole uterus is felt.

To palpate the right ovary, two fingers of the right hand are passed into the vagina to the patient's right. Pressure is made in the same direction by the left hand on the abdomen. This usually forces the right ovary between the fingers. One should be able to feel the entire surface and all the borders of the ovary. In order to feel the left ovary, use the left hand in the vagina and the right one in the abdomen. The tubes

are palpated in the same manner as described for the ovaries. A normal tube usually cannot be palpated.

There has been considerable discussion as to which hand

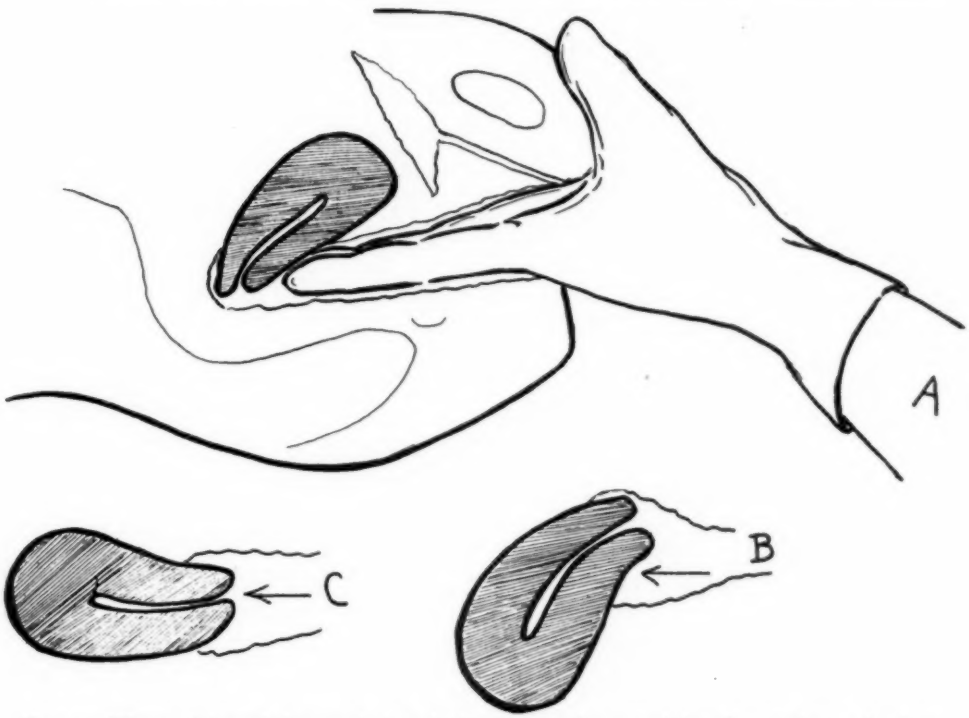


FIG. 12. The position of the cervix in relationship to the examining finger gives a lead as to the position of the uterus. A, striking anterior lip, uterus is probably in anterior position; B, striking posterior lip, uterus is probably in posterior position; C, striking external os, uterus is probably in the axis of the vagina.

to use in the vagina. It would only take a short time to convince anyone using only one hand, that they should use two hands, the right for the right side of the pelvis and the left for the left side. If only one hand is used in the vagina, it becomes necessary to twist oneself around into a very uncomfortable position to feel the opposite ovary. By following this procedure, using the right hand in the vagina for the right side and the left hand for the left side, fewer masses will be missed.

Rectovaginal-abdominal examinations should be performed more frequently in order to obtain a better concept of the size, shape and mobility of masses lateral to the uterus. One finger is placed in the rectum, one in the vagina and the other hand is

placed on the abdomen. There is a marked difference in "feel" compared to having the fingers only in the vagina.

If the vaginal examination shows a soft, cystic, movable, globular, non-sensitive swelling to the side of the uterus, it is probably an ovarian or parovarian cyst. When it is hard, solid, movable, globular, non-sensitive it is usually a fibroid or a solid ovarian tumor. If it is a firm, fairly hard, fixed, tender swelling it is usually inflammatory, either a parametric exudate or tubular inflammation.

The position of the cervix is very important particularly as an index of the probable position of the fundus.

If on inserting the examining finger it strikes the posterior lip of the cervix, the uterus is probably in a posterior position. If the vaginal finger strikes the anterior lip of the cervix, the chances are that the uterus is in anteflexion. If on inserting the finger it strikes the external os, the uterus is probably in the axis of the vagina (Fig. 12).

The consistency of the cervix is very important. *Do not be fooled by the rough "feel" of a cervix which has been repaired.* No matter what type of operation is performed on the cervix, it is surprising how lacerated that cervix feels to the examining finger; and yet on visual examination an intact mucous membrane is found and no erosion or pathological process is seen. Visually examine every cervix that is felt. If the cervix feels hard, look out for carcinoma. Normally it should feel like the end of the nose. If it feels soft and velvety, it is probably eroded. A rough feel usually means cysts or possibly polyps.

A lacerated cervix can frequently be felt and not seen. It is not unusual to make a vaginal examination in a patient two, three or four months postpartum and find that the cervix feels badly lacerated and yet on visual examination it looks normal. This is because the mucous membrane has not been torn but the cervical musculature has had a submucous laceration.

The mobility of the cervix is very important; as also is the presence or absence of tenderness. Any fixation of the

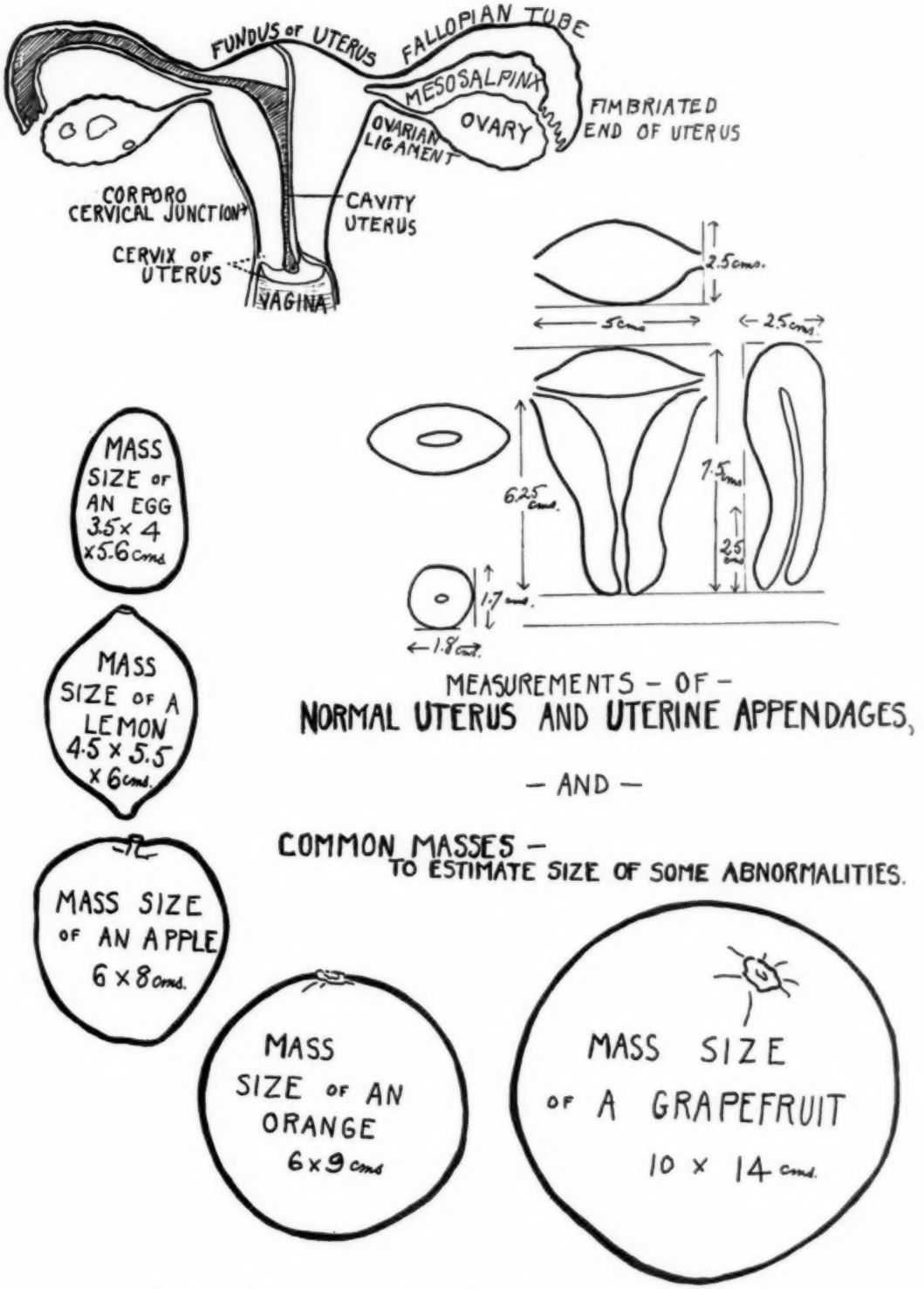


FIG. 13. Chart showing size of uterus and masses. (*After Holden.*)

cervix is significant, it means inflammation, new growth or endometriosis.

A description of the examination of the uterus should cover its size, shape, position, surface, mobility, consistency and tenderness. The same things should be described about the ovary.

When describing the tubes, remember a normal tube cannot be palpated. A mass in the region of the tube which is cord-like is a salpingitis. If it is sausage shaped it is a pyosalpinx. If it is globular and sausage shaped and fairly fixed, it is a tubo-ovarian abscess. In the acute cases of tubal infection, a pelvic peritonitis is usually present. Therefore, there will be a splinting of the vault of vagina by exudate. This splinting plus the rigidity of the lower abdominal muscles, makes it impossible to diagnose the exact pathology present at the first examination. In these acute cases it is usually necessary to wait until some of the exudate is absorbed and some of the acute symptoms subside, then the localized masses can be better palpated and its pathology properly evaluated.

In describing masses felt to one side of the uterus, give its exact size in centimeters, location, relationship to the uterus, feel of surface, mobility, tenderness and consistency. Do not use store terms such as, size of an egg, lemon, grapefruit. To prevent this and to facilitate the description of pelvic masses a chart (Fig. 13) should be on the wall in the examining room in such a position that the examiner would be looking at the chart while making the examination.

In elderly women with pelvic masses, the *x*-ray—barium enema—should be used more frequently to eliminate bowel pathology. The proctoscope and sigmoidoscope have been too long neglected by the gynecologist as an aid to pelvic diagnosis.

Visual Examination. A well lubricated bivalve speculum should be used. When the speculum is introduced for the examination of semen or trichomonas, no lubricant is used. When introducing the speculum put it in closed and at an angle of 45° to the perineum (Fig. 14A). One finger is pressed

downward on the perineum to give the necessary room (Fig. 14C). After the speculum is in the vagina a short distance, it is rotated to the transverse axis (Fig. 14B-B).

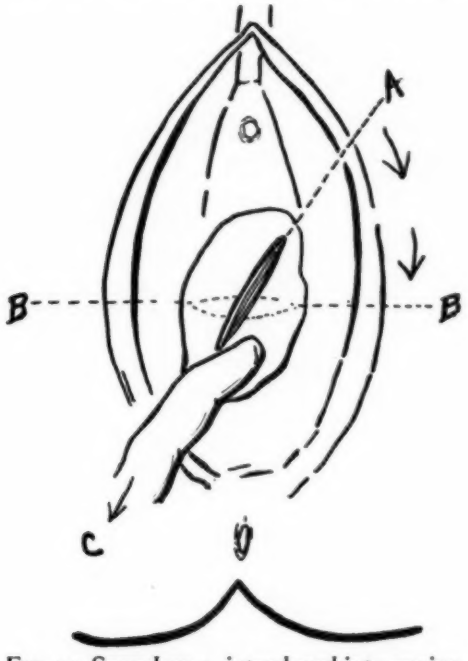


FIG. 14. Speculum A, introduced into vagina at an angle of 45 degrees; C, finger of left hand depressing perineum; arrow shows rotation of speculum to transverse axis, B-B.

When inspecting the cervix, observe if it is smooth, firm, swollen or hypertrophied. Is the external orifice a slight dimple or a transverse slit? Is there a laceration of the external orifice? If lacerated, is there gaping? Lacerations occur mostly at the sides; very rarely does one find lacerations in the midline. Is an erosion present? Erosions are not ulcerations. All ulcerations must be biopsied as they are suspicious of possible carcinoma.

Attempt if possible, to give the size of the cervix in centimeters. There is at present a new method of examining the cervix by four power magnification but its value is still problematical.

The discharge from the cervix should be described. Is it light or profuse, glairy or clear, mucus or mucopurulent?

Chapter II

PREOPERATIVE HOSPITAL ROUTINE; PREPARATION OF THE PATIENT; POSTOPERATIVE HOSPITAL ROUTINE

No operation should be performed on any patient without a complete laboratory work-up, except in an emergency. Then, only the basic laboratory work should be done, e.g. a urine and blood count, etc.

In most public institutions, except in emergencies, it is advisable to keep the patients in the hospital at least seventy-two hours before operation. These patients are usually overworked, underfed and undernourished and a seventy-two hour rest is of distinct benefit. On any very active service, if the patients are admitted one night and operated upon the next day, unexpected pathology will be encountered. One cannot tell just what pathology to expect until a patient has been under observation for at least forty-eight hours. What appears to be a simple case on admission may, at the end of twenty-four to forty-eight hours, develop a rising temperature with a marked change in her pelvic pathology.

Preparation of the Field for Operation. The preparation of the field for the operation should be simple and should cause as little injury to the skin as possible.

The field should be carefully shaved, including the abdominal wall, vulva and perineum. It is far better to shave too large than too small a field. In gynecological cases all patients should be prepared vaginally as well as abdominally even though a vaginal operation may not be contemplated. One cannot foretell when, in the course of an operation, it may be necessary to enter the vagina.

The night before the operation the abdomen is shaved and scrubbed with soap and water. Sometimes it is necessary to use benzine to clean out the umbilicus, but occasionally this may cause skin burns. Following mechanical cleansing the abdomen is washed off with 70 per cent alcohol and clean, dry,

sterile dressing applied which is not removed until the patient is in the operating room.

In the vaginal preparation do not use alcohol, benzine or iodine as the patient will complain bitterly of burning. At this time the use of a lysol douche is usually sufficient.

The patient must have a good night's rest before the operation. Give a sedative if necessary. One hour before the operation, the patient should receive morphine grain ¼ and atropine grain $\frac{1}{150}$ (use tablets, not solution)* unless the order is countermanded by the anesthetist. This makes the anesthesia smoother and less of the anesthetic agent is necessary.

All foreign bodies of any kind should be removed from the patient's mouth and hands before she goes to the operating room. It is dangerous for a patient on the operating table to have a loose dental plate rattling around in her mouth. Sometimes such objects are swallowed or the plate or bridge may enter the trachea.

When specific preparations for definite operations are necessary they will be discussed with that particular operative procedure.

Preoperative Diet and Catharsis. No cathartics are ordered for patients preoperatively as a routine. Brisk preoperative catharsis which for years was the routine for all patients has gradually been eliminated. It has been found not only unnecessary but it seems to increase the postoperative gas pains and ketosis. The patient should receive an enema the day before and a second one early on the morning of the operation. In cases where avertin is to be used, the morning enema should be omitted. If it is given the rectum will discharge the avertin and the effect of the drug will be lost.

In some specific instances such as umbilical hernia, ventral hernia, etc., in obese patients, it is recommended that they be moderately starved for two or three weeks before the operation. The patient's intake is limited so that she loses at least ten pounds in weight. This is to prevent a postoperative evul-

* The use of tablets instead of solutions of morphine diminishes the chances of error in the dosage.

sion of the abdominal wound due to distention. The overweight patient postoperatively may not have enough room in the abdominal cavity for her distended intestine or stomach unless this precaution is taken. As Dr. George D. Stewart used to say "they have lost their right to domicile." These cases are given cathartics three to four days before the operation but none immediately before.

During the patient's preoperative stay in the hospital, as part of her preparation, she should be made to drink large quantities of fluids and eat a moderate amount of carbohydrates, as candy and other foods of that type which have a high caloric content with a low residue. This will tend to prevent postoperative shock and ketosis.

The patient should receive a soft diet the night before and no breakfast the morning of the operation. If, however, the operation is to be in the afternoon, she is given a light breakfast.

When emergency cases are admitted, the house surgeon, if he thinks urgent surgery is necessary, should order the patient's abdomen and vagina prepared, as soon as the Attending is notified of the admission. This emergency preparation consists of shaving the abdomen as for a dilatation and curettage and laparotomy. Preoperative low enemas in emergency cases are optional. Do not give an enema to a suspected ruptured ectopic pregnancy.

Preparation of the Vagina—under Anesthesia

The vagina and surrounding areas are painted with mercurochrome (2 per cent mercurochrome in acetone and alcohol). This is done in the anesthesia room so that the vagina is rendered sterile.

The patient is catheterized at this time. All patients should be catheterized immediately before entering the operating room. See to it that urine is obtained at catheterization and that the bladder is empty. A simple hysterectomy can be a most difficult operation when there is a full bladder crowding the pelvis.

Following catheterization instill ½ oz. of either 10 per cent argyrol solution or ¼ per cent protargol solution into

the bladder. This will minimize the possibility of a subsequent cystitis.

Chemical Skin Sterilization

The preliminary preparation of the operative field has not changed materially in the last decade. However, there has been a constant search for the ideal chemical "skin sterilizer." The requirements necessary for the ideal chemical skin disinfectant should be:

*1. It must have the ability to kill rapidly the usual contaminating skin organisms. This property should last for several hours.

2. It must be able to dissolve the skin debris and penetrate the surface of the skin.

3. It must dry fairly rapidly so as not to delay the operative procedure.

4. It must have a high color index so that there shall be no question as to the extent of the sterilized area.

5. It must be non-toxic so as not to interfere with the normal healing process.

6. Its application should be painless, particularly for local anesthesia operation.

7. It must be non-irritating.

8. It must be stable.

9. It must be inexpensive.

The "skin sterilizers" most frequently used are tincture of iodine 3½ and 7 per cent; picric acid 5 and 2½ per cent in 95 per cent alcohol; and mercurochrome 2 per cent in acetone and alcohol (mercurochrome 2 grams dissolved in 35 c.c. of distilled water, containing 0.1 gram sodium carbonate, then adding while stirring 55 c.c. of 95 per cent alcohol U.S.P. and 10 c.c. of acetone U.S.P.).

Precautions

Tincture of Iodine. 1. Forms a blotchy surface with marginal concentration and skin burns are frequent if iodine is not removed with alcohol.

2. If iodine is allowed to drip around the patient's body and she lies on iodine soaked towels a burn on the back frequently occurs.

3. A large number of people are sensitive to 7 per cent iodine; when iodine is allowed to stand the alcohol evaporates and the solution becomes stronger. This also causes frequent burns.

*Falk, H. C. Operating Room Procedure. New York, G. P. Putnam's Sons, 1934.

4. No towels, sheets or linens are to come in contact with the iodinized area while wet. If they do the iodinized sheet causes a burn where it remains in contact with the skin.

5. Cover all iodinized skin with towels so that the intestines do not come in contact with it as the injured serosa may lead to intraabdominal adhesions.

Picric Acid. 1. One occasionally finds people very susceptible to picric acid with a resulting generalized dermatitis.

2. All cloths saturated with picric acid and allowed to dry are highly inflammable.

3. Picric acid does not act well in the presence of free albumin (blood, raw tissue) therefore it should not be used in open wounds.

4. The solution stains bed clothes and linens for days after its application.

Mercurochrome. 1. Very expensive when compared with other solutions.

2. Red color rather difficult to remove but does not come off on to clothes, once it has dried.

3. The solution is not difficult to prepare but must be properly prepared in order to get best results.*

General Postoperative Orders. The patient is to receive nothing by mouth until nausea and vomiting cease. When fluids are begun, they should be given in dram doses of a warm solution (warm tea for example) and should be increased gradually.

Fowler's position should be used on all pelvic cases after the patient returns to consciousness with the exception of ectopics in shock or spinal anesthesia cases. The head of the bed is not to be raised more than 12 inches except on a special order. In all drainage cases the position is to be maintained until all drains are removed.

* Discoloration from mercurochrome is removed:

1. From the skin, if stain is fresh by the use of acidulated alcohol (2 per cent hydrochloric acid in alcohol).

2. From colorless cotton or linen cloth:

Mix one volume of Labarraque's Solution (chlorinated soda) with four volumes of water. Immerse the stained fabric in this mixture and allow to soak for about two minutes. Without removing the fabric, add one volume of 5 per cent acetic acid (approximately the strength of vinegar) and mix thoroughly with a stick. The stain disappears rapidly and should be completely removed in one minute.

Falk, H. C. Operating Room Procedure. New York, G. P. Putnam's Sons, 1934.

During the early postoperative hours the patient receives 6 ounces of tap water by rectum every six hours, to be retained until fluids are taken by mouth. This fluid is to be begun as soon as the patient returns to her bed, except in avertin cases when the patient should receive 16 ounces of coffee, this to be followed by tap water 6 ounces every six hours which is to be retained. The rectal fluid is to be discontinued when the patient takes sufficient fluids by mouth.

No rectal fluids are to be given to patients:

1. Suspected of injury to the intestinal tract,
2. Where known injury to the lower intestinal tract has occurred,
3. In complete lacerations of the perineum, or
4. In ordinary perineorrhaphies, except by special order.

There must be a record on the chart of the fluid intake of all postoperative cases until the patient takes fluids freely. Every effort must be made to maintain a 3000 c.c. fluid intake per day using rectal tap water, hypodermoclysis and infusions if necessary.

The patient is to get morphine sulphate gr. $\frac{1}{4}$ with atropine gr. $\frac{1}{150}$ every six hours, for the first twenty-four hours s.o.s. Solutions for the hypodermic injections are to be freshly prepared from tablets. To prevent the constipating effect of morphine when used postoperatively, some men give a hypodermic injection of 1 c.c. surgical pituitrin with every one-fourth grain of morphine, but we have never used it as a routine procedure.

Hypodermoclysis shall be given as ordered when the patient's fluid intake is too low. Infusions are to be used only on special order. Infusions and transfusions shall be given when necessary by special order only.

When the patient is returned to bed she should be placed on her side to prevent pulmonary complications by aspiration. Her blood pressure should be taken and if the pressure is below 100, the interne must remain with that patient until the blood pressure returns to 100. These patients should be placed in reverse Fowler's position and given a hypodermoclysis of saline, (*never use glucose solution as a hypodermoclysis as it*

may cause a slough). An infusion or a hypodermic of caffeine sodium benzoate gr. 7½, camphor in oil 1 c.c. or adrenalin ½ to 1 c.c. subcutaneously may be necessary. Retention of body heat is effected by blankets and hot water bags, but do not burn the patient, remember she is unconscious. Where there has been no vaginal surgery, the Elliott apparatus may be employed to raise the body temperature. If the blood pressure is below 80 or if the pressure does not respond in fifteen to thirty minutes to the methods of stimulation used, the surgeon should be notified and he must respond immediately.

Cathartics are not used as routine postoperative procedures. The patient is to receive a soap-suds enema on the second day after the operation. Where indicated, a mild cathartic can be used as: milk of magnesia or, mineral oil in tablespoonful doses, fluid extract of cascara sagrada drams 1 or pulverized cascara sagrada grains X.

In those cases where the appendix has been removed there has been interference with the continuity of the gastrointestinal tract, therefore cathartics should not be used as readily and as frequently as otherwise. Catharsis in these cases is delayed for twenty-four to forty-eight hours.

All clean cases should sit up in bed on the eleventh or twelfth day, should be out of bed on the thirteenth day and may be discharged as soon as they can walk and take care of themselves. Infected cases are not to get out of bed except on special order.

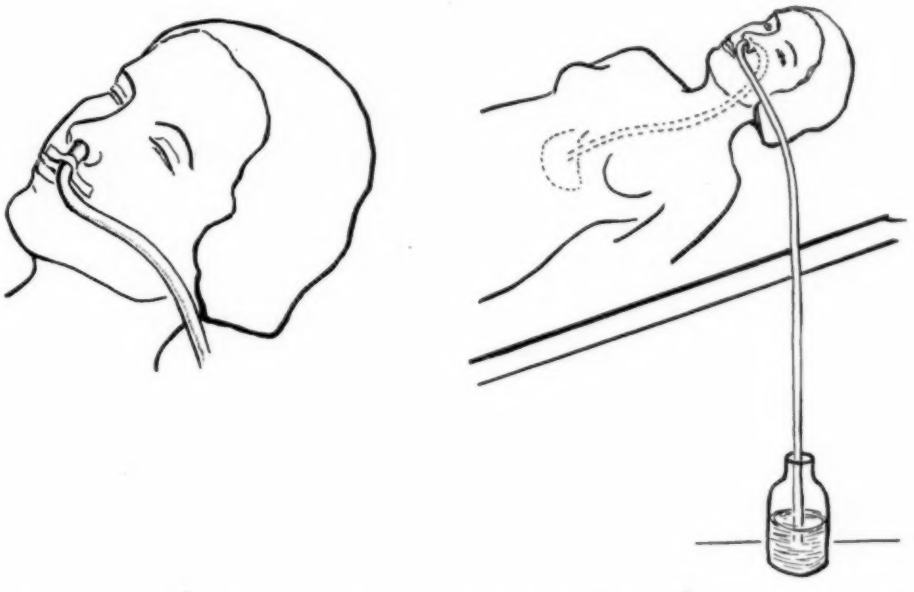
Treatment of Postoperative Complications. The treatment of postoperative complications consists of (1) prophalaxis and (2) active early treatment.

Do not wait to start active treatment until the patient has developed persistant vomiting or marked distention. Begin the treatment early and it will frequently be found that the expected complication will not develop.

Routine Orders for Postoperative Complications

1. *Vomiting.* If the patient vomits more than usual or for more than twelve hours after her operation, she should have a

gastric lavage using the stomach tube, or the patient should drink two glasses of warm, dilute milk of magnesia (one tablespoonful to one glass of water) and be allowed to vomit this



A B

FIG. 15. A, a piece of adhesive placed over the tube close to its emergence from the nose will help to keep the tube in place; B, a duodenal tube through the nose into the stomach, showing the distal end of the tube below the level of the stomach so that siphonage can be carried out successfully.

mixture. The alkali helps dissolve the mucus in the stomach and the fluid gives her something to vomit. Following this, the patient should receive a dose of morphine gr. ¼. She will then go to sleep and when she awakens she is usually comfortable and the period of nausea and vomiting is past.

If the patient continues to vomit after this, insert a Levine tube and lavage the stomach. This requires care, time and patience. To do this properly, the patient may drink water by mouth and allow the water to siphon back through the Levine tube, thus the patient washes her own stomach, or the water can be withdrawn with a 20 c.c. syringe. *The distal end of the Levine tube must be lower than the stomach if gravity siphonage is to work* (Fig. 15B). In lavaging the stomach with a Levine tube, see to it that at least as much fluid is withdrawn as is inserted.

Measure both quantities. In that way the patient cannot be left with a dilated stomach filled with fluid. The Levine tube must not be left open and draining continuously, as the patient will readily dehydrate herself. Patients must not be allowed to lose more fluids than they take as they lose very necessary chlorides. Patients must retain at least 3000 c.c. of fluid a day. The Levine tube should be "closed off" during definite periods of time, one-half hour every hour. A specified time for closure of the tube should be mentioned on the orders when the Levine tube is inserted.

When it is necessary or desirable to start the gastrointestinal tract functioning, milk of magnesia oz. 1, castor oil oz. 1 or magnesium sulphate oz. 1 in one-half glass of water may be inserted through the Levine tube.

2. *Acute Gastric Dilatation.* It is very important to diagnose this condition early. These patients have respiratory distress and cardiac embarrassment which makes them feel very uncomfortable. When a patient has upper abdominal distention and the lower abdomen is moderately flat, think of gastric dilatation. One should suspect this condition in every patient where there is vomiting and upper abdominal distention of an undue nature. Some of these patients will not vomit, but will simply spillover, e.g. they will bring up mouthfuls of brown foul smelling fluids. In these cases the stomach tube should be passed immediately and the stomach washed out with milk of magnesia oz. 1 to 200 c.c. of water or a 5 per cent sodium bicarbonate solution. Use this fluid freely until the return flow is absolutely clear. This may require an hour or more. Use the syringe for suction and have the patient drink the fluid by mouth.

Placing the patient in Fowler's position helps the stomach to rid itself of the gas while placing the patient in reverse Fowler's helps drain the fluid. The Fowler's position should therefore be used for the gas and the duodenal tube siphonage for the fluid. In some cases it may be necessary to have a continuous lavage in order to keep the fluid and gas from accumulating. There are machines on the market which maintain a

continuous hydraulic suction on the duodenal tube. A special nurse is necessary for all patients with acute gastric dilatation. If the gastric dilatation does not improve, the patient probably has some other abdominal complication, possibly an associated peritonitis.

These patients should also receive fluid by rectum, 6 ounces tap water every six hours or by hypodermoclysis. Fluids can be given through the duodenal tube if desired, in which case it is necessary to see to it that the tube is in the duodenum and not in the stomach; then the Murphy method of giving fluids can be used giving sixty drops a minute into the duodenal tube.

3. *Abdominal Distention.* This is a frequent postoperative complication and may be very distressing. It is a form of ileus, probably due to the handling of the intestines during surgical procedures. It is essential in all cases of abdominal distention to rule out the possibility of a developing peritonitis.

Prophylaxis. Be very gentle in handling the intestines; do not disturb them more than is absolutely necessary.

Active Treatment. At the first sign of distention these patients should all have a rectal tube inserted and a low soap-suds enema given. If the soap-suds enema does not work, an "M.G.W." enema consisting of 2 oz. each of magnesium sulphate, glycerine and water, is frequently of value.

The Harris drip is very efficacious, particularly in lower abdominal distention. The success of the Harris drip depends upon the careful observation of details (Fig. 16).

1. The can, which is preferable to a rubber bag, should be about one-half full of warm fluid at about 105° to 106°F. The temperature of the water is maintained by an electric bulb in the can.

2. The bottom of the can should not be more than one foot above the level of the bed and the top of the fluid should be 4 inches above the level of the rectum.

3. The rubber tubing from the can should be of fairly large size and should be connected with a large rectal tube.

4. Before inserting, the rectal tube should be well lubricated and a small amount of fluid allowed to escape, so as to fill the tube and eliminate

any air pockets. While the fluid is running the rectal tube is inserted for a distance of about 4 inches.

5. The fluid in the rectum will promptly reach the level of the fluid in

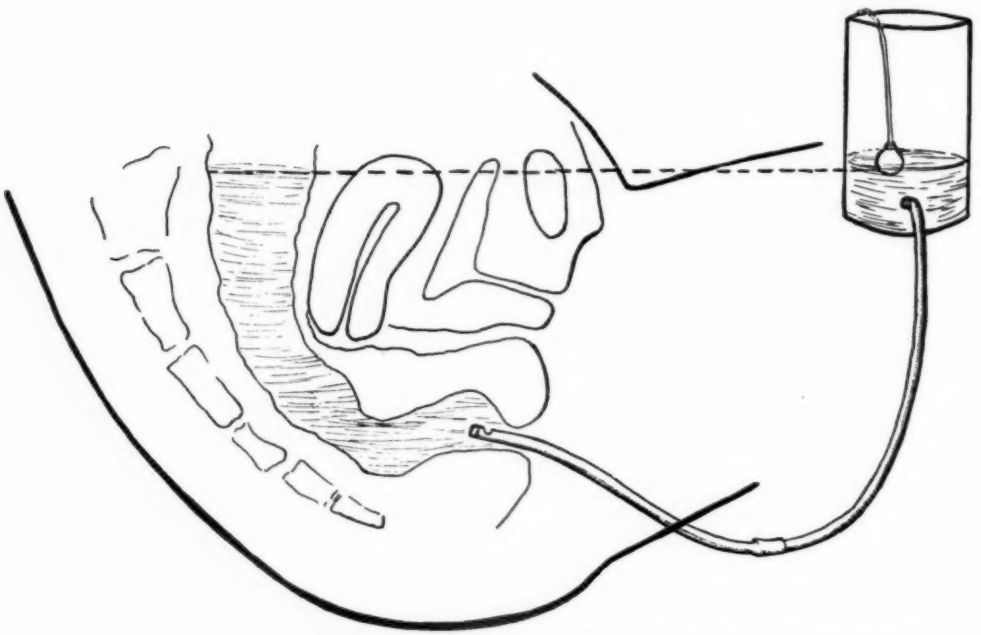


FIG. 16. Harris drip, showing can, with light, slightly above level of rectum. Can should be lowered to the floor every fifteen minutes.

the can and there will be a free exchange of gasses and fecal matter with a moderate absorption of fluid.

6. Every fifteen minutes the can should be lowered to the floor to allow the fluid in the rectum to escape.

A Harris drip that is working properly always has the fluid stained with fecal matter.

Colonic irrigations may be used with or without a preliminary course of pituitrin, consisting of 1 c.c. pituitrin given every three-quarters of an hour for three doses; one-half hour after the last dose the patient receives a colonic irrigation.

The application of heat to the abdomen is a very good procedure, and resort to turpentine stupes may sometimes be necessary. If this is indicated all abdominal dressings should be removed except those immediately covering the wound, and the turpentine stupes applied directly to the abdominal wall.

In those cases where there has been no interference with the continuity of the gastrointestinal tract, such cathartics as milk of magnesia, mineral oil or cascara sagrada, may be used with impunity. In those cases where there has been interference with the continuity, such as appendectomy, intestinal resection, intestinal injury, etc., pitressin should be used.

In all abdominal cases with general peritonitis at the time of the operation pitressin should be used. It should be begun as soon as the patient leaves the table, one ampule every four hours for forty-eight hours. The duodenal tube should also be inserted as soon as the patient is conscious and gastric lavage with milk of magnesia water begun almost immediately.

Spinal anesthesia in the treatment of abdominal distention is not recommended as a routine procedure although it has been used in cases with marked persistant distention. It paralyzes the inhibitors and allows the unopposed action of vagus stimulation thus producing marked peristalsis. If the spinal anesthesia does not relieve the distention, a mechanical obstruction or general peritonitis must be considered and the patient should be ready for operation under the same anesthesia.

4. *Urinary Retention.* In gynecological cases it is very essential to watch the urinary bladder very closely. If the patient has had a hysterectomy, immediate catheterization is advisable to be sure the bladder has not been injured. Injury to the bladder is not serious if recognized at once and immediately repaired with a double layer of fine chromic sutured.

Routinely the gynecological patient should be catheterized about ten hours postoperatively. If she becomes too uncomfortable, the catheterization may be done before the ten hours have elapsed. Argyrol or protargol should be instilled into the bladder after every catheterization. Do everything possible to avoid catheterization. If the patient has been catheterized and then begins to void, she should be catheterized once a day after voiding until she completely empties her bladder as it is not unusual to have 3 to 4 ounces of residual urine after voiding. It is this residual urine which seems to cause the

damage. Catheterize until there is no residual urine after voiding and instill argyrol or protargol after every catheterization.

Pulmonary Complications: Bronchitis, Pneumonia, Atelectasis, Collapse

In spite of all the theories that have been advances regarding postoperative pulmonary complications, the aspiration theory is the one most generally accepted. If this be true, then prophylaxis is more important than therapy.

The prophylaxis can be divided into three parts:

1. Preoperative preparation of the gastrointestinal tract and proper cleansing of the mouth and teeth,
2. Well given anesthesia, and
3. Postoperative care.

Preparation of the Mouth. The teeth and gums should be examined by a dentist and all carious teeth either filled or extracted. The regular use of a tooth brush should be insisted upon.

Preparation of the Gastrointestinal Tract. Except in emergencies, no patient should be operated upon whose stomach is not empty.

Well Given Anesthetic. This cannot be over emphasized. While the proper premedication such as atropine, scopolamine, etc. tends to dry the throat, the depth of anesthesia with its proper maintainance allows a quick return of the reflexes and thus prevents mucus from collecting in the throat. A well conducted anesthesia also diminishes postoperative vomiting.

Postoperative Care. Patients should be well protected from all draughts to and from the Operating Room. All wet clothing should be removed in the Operating Room and replaced with warm, dry clothing. Postoperatively most patients incorrectly are allowed to lie on their back for their recovering hours. This allows mucus and other fluids to collect in the throat and thus are readily aspirated.

All patients, if the operation does not contraindicate it, should be placed on their sides until they have recovered consciousness.

ACTIVE TREATMENT. If a patient develops a postoperative cough, a medical consultation should be called immediately, and the following orders should be given:

1. Fowler's position,
2. Pneumonia jacket,
3. Sodium citrate gr. xx every four hours for forty-eight hours, and
4. Some form of a cough sedative, as codeine cough mixture.

Occasionally after operation, patients develop atelectasis (deflation of the alveolar tissue but not of the bronchi) or collapse (deflation of alveolar tissue and bronchi) of one or more lobes. This usually manifests itself within the first four postoperative days. Clinically it manifests itself by a marked rise in the pulse rate out of all proportion to the pulmonary symptoms, cough, slight dyspnea or the temperature.

The generally accepted theory as to the etiology is that one of the large bronchi becomes plugged with a mass of tenacious mucus. *X*-ray picture of the chest will usually establish the diagnosis. In taking the radiogram it is advisable to have the patient moved from the bed to the stretcher and back again as this seems to help dislodge the plug of mucus. Having the patient cough and striking her on the back at the same time may also help to dislodge the mucus plug. The patient should be encouraged to change her position frequently. If the pulse rate becomes very rapid and the patient is quite dyspneic, oxygen should be given through a nasal catheter. If the collapse is extensive and is not relieved by these methods a bronchoscopy may have to be done in order to dislodge the mucus plug.

What shall be done with patients in acute postoperative surgical shock?

1. Keep the patient's body warm with external heat. Wrap the patient well in blankets and apply hot water bottles but not too hot as the patient may be unconscious. The use of the Elliott Apparatus with

the bag in the vagina may help maintain bodily heat. It, of course, should not be used in those cases that have had vaginal operations.

2. Elevate the foot of the bed.

3. Give saline by hypodermoclysis and infusions.

4. These patients usually perspire very freely. Have their clothes changed frequently. Do not allow a patient in shock to lie in wet underclothing as this increases shock and predisposes to pulmonary complications.

5. Type the patient for a blood transfusion and have donors available. Do not wait too long before giving a transfusion.

6. Place the patient on the "Serious List."

7. Unless there is a distinct contraindication give the patient enough morphine to quiet her.

8. Take the patient's blood pressure at least every hour; every half hour if necessary.

9. The patient may receive hypodermic stimulation: caffeine sodium benzoate gr. v, camphor in oil 1 c.c., adrenalin 1 c.c. intramuscularly and strychnine gr. $\frac{1}{120}$, these are all advised but as a rule are of little value.

10. When the patient is in shock, don't transport that patient from one ward to another or from the bed to a stretcher. If the patient must be transferred to a private room, transfer the bed with the patient.

11. Be absolutely sure the patient is not suffering from postoperative hemorrhage instead of surgical shock. To do this, have a hemoglobin and red blood cell count taken at frequent short intervals. In shock, owing to the drawing off of fluids from the circulation there is a hemoconcentration with a high red blood cell count and high hemoglobin, whereas in hemorrhage, the red blood count and hemoglobin drop.

The most important spot on the patient's face to palpate for shock is the nose and cheek. If the patient's nose and cheek are cold and clammy, that patient is usually in shock, unless she has just come in from the cold. A cold, clammy, sweating skin means shock. Hemorrhage usually shows itself by restlessness and air hunger. A patient who is suffering from hemorrhage rarely lies quietly, she is usually tossing around in bed.

Chapter III

ABORTION

The most frequent single condition seen on an active gynecological service is abortion. From the clinical standpoint it is necessary to regard every abortion, complete or incomplete, admitted to the hospital as an induced abortion. In studying 1000 consecutive cases of abortion an attempt was made to correlate the symptomatology with the pathology and it was found they could be classified into definite pathological groups. Following this classification it was possible to obtain a fairly accurate idea of the pathology one would expect to find in any given case by carefully studying the history, clinical course and the temperature chart of the patient.

Pathology of Abortion. When a patient loses the products of conception, there is a raw surface in the cavity of the uterus; this is a surgical wound. The cervix must dilate to allow the products of conception to be discharged, with the result that there are numerous small breaks in the cervical mucosa. These breaks in the continuity of the cervix become infected with the streptococci and staphylococci in the racemose glands of the cervix and these organisms extend upward into the uterine cavity to the surgical wound by direct extension or along the subepithelial lymphatics.

Bacteriology of the Vagina and Uterus. Normally the cavity of the uterus is sterile. A virgin vagina contains large numbers of Doederlein bacilli but no pathogenic organisms. The cervix before sex trauma is also sterile. With the advent of sex trauma, the cervical canal which is lined with compound racemose glands, becomes infected with the usual pathogenic organisms, chief of which are the streptococci and staphylococci. These organisms are present in different types, virulence and numbers in the cervix following exposure and the individual patient has a variable resistance to the organisms which she harbors.

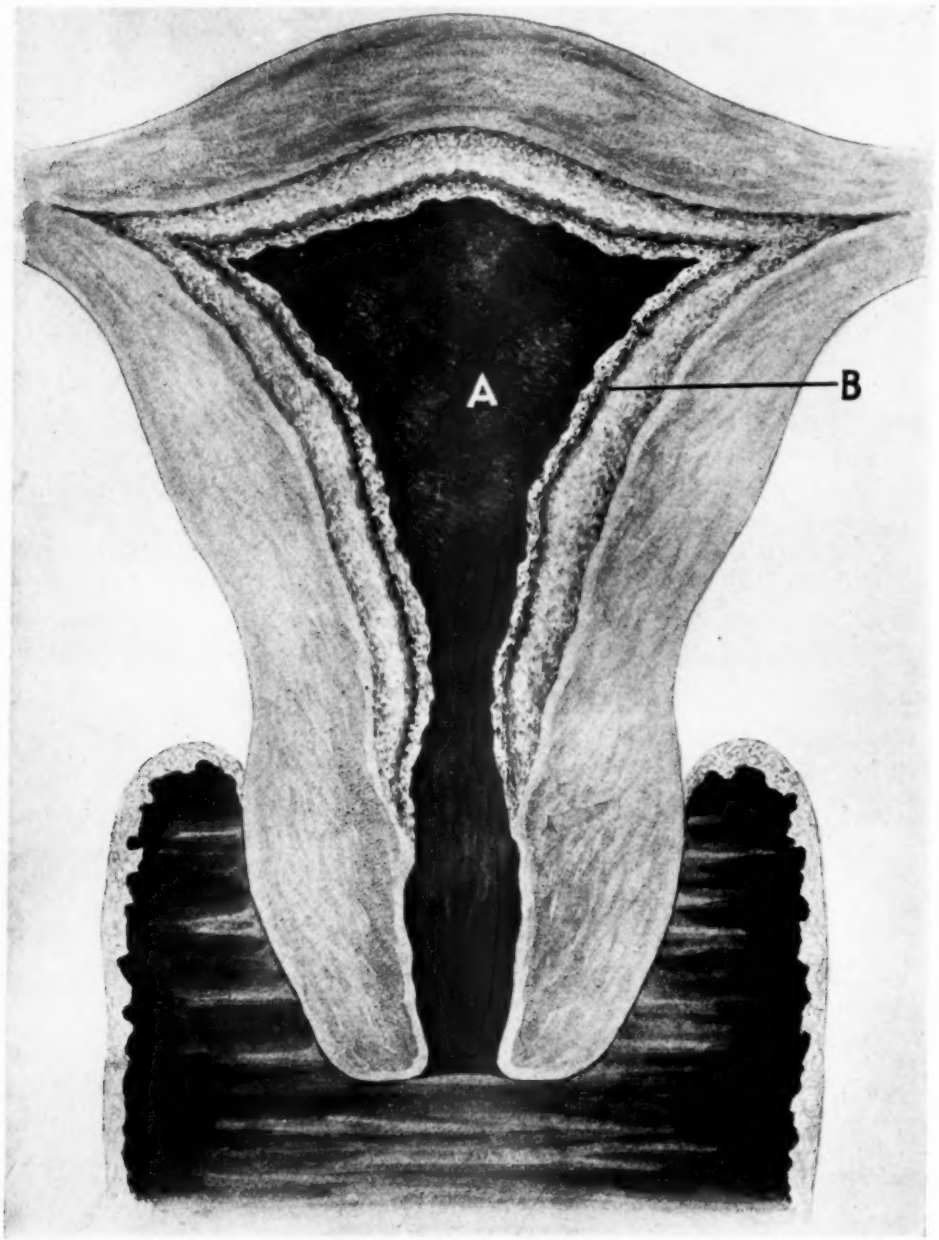


FIG. 17. Type 1 postabortal infection; infection limited to the cavity of the uterus, A, infected uterine contents; B, leucocytic layer of Bumm.

The forms of postabortal infections of the uterus may be divided into *seven groups or types.*

Type I. Infection Limited to the Uterine Cavity. In this type the cavity of the uterus is lined with an infected membrane. It may or may not contain pieces of placental tissue. Underneath this infected tissue a leucocytic wall is thrown up, known as the leucocytic layer of Bumm, (Fig. 17) which prevents the infection from extending beyond the uterine cavity into the broad ligament and into the general system. These patients may or may not have a temperature. Cultures taken from the uterine cavity with Little's technique in 50 consecutive afebrile abortions gave positive cultures in 23 instances, streptococci, staphylococci and in one case, an aeorogenous capsulatus was found. Type I postabortal infections consist of those cases in which the infection has not extended beyond the uterine cavity.

Type II. Parametritis, Etc. If the virulence of the infecting agent is greater than the resistance of the individual, the infection goes beyond the layer of Bumm through the lymphatics and out into the broad ligament (Fig. 18). The infection primarily goes through the lymphatics and later involves all the cellular tissue of the broad ligament. When this occurs the cellular tissue and the lymphatics in the broad ligament act as a second barrier with the development of a parametritis.

Accompanying the involvement of the cellular tissue of the broad ligament there is marked exudate of leucocytes and serum between its layers. This causes the normally thin broad ligament to become markedly swollen (Fig. 19). This exudate between the layers of the broad ligament may go forward towards the bladder or even in front of the bladder, or it may go backward toward the rectum or around the rectum (Fig. 20). The peritoneum of the cul-de-sac of Douglas may be pushed upward so as to obliterate the sac completely. The cul-de-sac exudate around the rectum may bulge into the vagina (Fig. 21). One must always remember that this exudate is entirely limited to the extraperitoneal structures. The exudate

FIG. 18. Type II postabortal infection; arrows show direction in which the infection travels into the broad ligament and out into the parametrial tissues. (*Modified from Polak.*)

is separated from the peritoneal cavity by the peritoneum itself which may become thickened and edematous (Polak's "peritoneal edema") or there may be some exudate on the

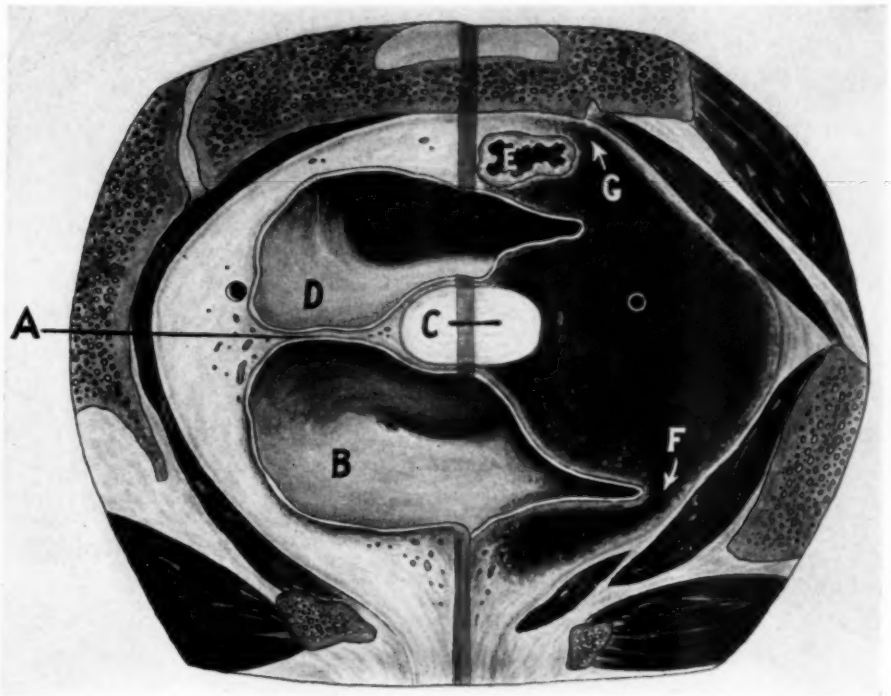


FIG. 19. Type II postabortal infection, right side; A, normal broad ligament; B, bladder; C, cervix; D, cul-de-sac; E, rectum; Left side, cellulitis between the layers of the broad ligament; F, extending anteriorly and deforming the bladder; G, extending posteriorly around rectum and cul-de-sac. (*Modified after Crossen.*)

abdominal side of the peritoneum. Occasionally, though very rarely, small pustular blebs may form underneath the peritoneum and rupture into the peritoneal cavity, causing a generalized peritonitis. This is infrequent for as a rule the Type II form always remains extraperitoneal.

These patients all have a cellulitis of the broad ligament, which may break down and form an abscess between the layers of the broad ligament. This abscess is extraperitoneal and may be drained through the vagina by a cul-de-sac puncture or above Poupart's ligament without entering the peritoneal cavity (Fig 22). Occasionally the infection, particularly that

part surrounding the rectum, may go upward and form a lumbar or perinephritic abscess, but it still remains extraperitoneal.

The Type II form of infection includes parametritis, peri-

FIG. 20. Type II postabortal infection; transverse section of the pelvis at the level of the internal os, showing exudate in the parametrium; A, cervix; B, ureters with catheters extending into C, the bladder; D, the rectum; E, posterior cul-de-sac, partially obliterated as a result of exudate, X, cellulitis in the parametrium.

metritis, prevesical abscess, abscess of the broad ligament, lumbar abscess, perinephritic abscess. Any of these abscesses may rupture externally, into a viscus, bladder, rectum, etc., or into the peritoneal cavity.

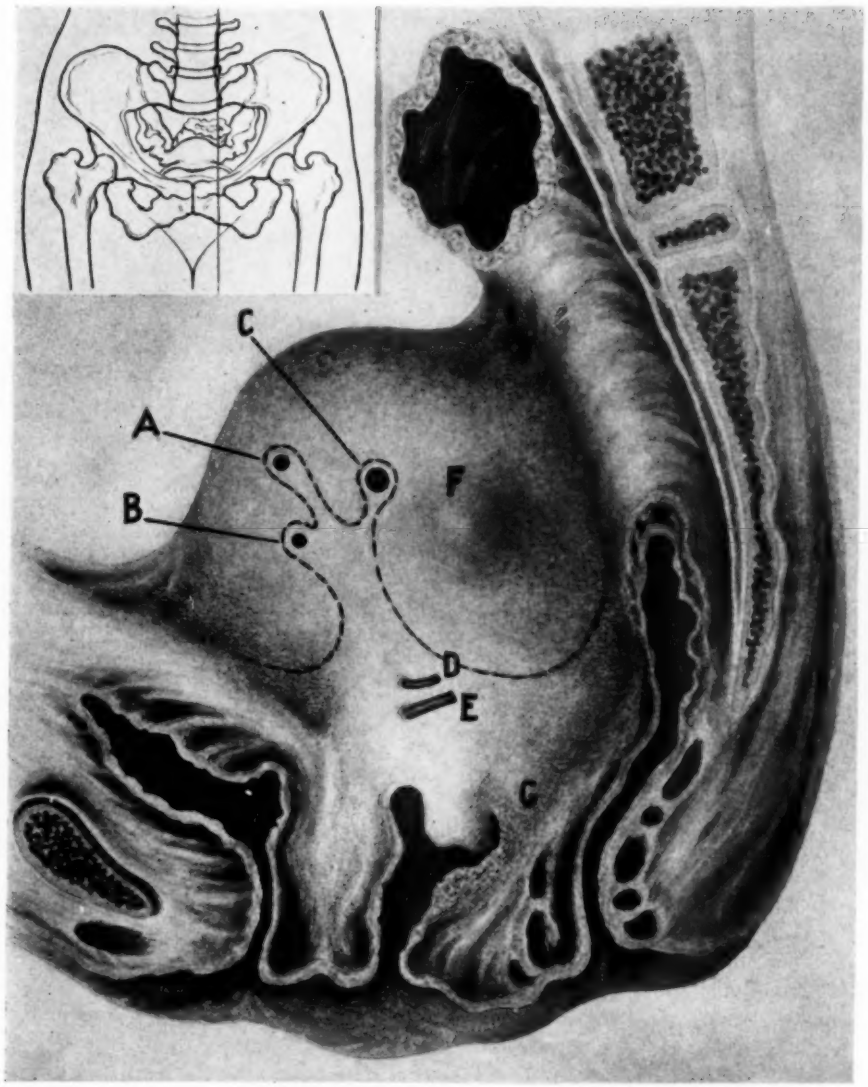


FIG. 21. Type II postabortal infection; sagittal section through the pelvis just to left of uterus. Dotted line shows the outline of the normal broad ligament and cul-de-sac with cross section of A, Fallopian tube; B, round ligament of the uterus; C, round ligament of the ovary; D, uterine artery; E, ureter; F, exudate between the layers of the broad ligament pushing the peritoneum upward and obliterating the cul-de-sac; G, exudate in the rectovaginal septum bulging into the vagina.

TYPE III. THROMBOPHLEBITIS. Thrombophlebitis develops in one of two ways. It is either the result of a direct extension of the infection from the large sinuses in the uterus, going

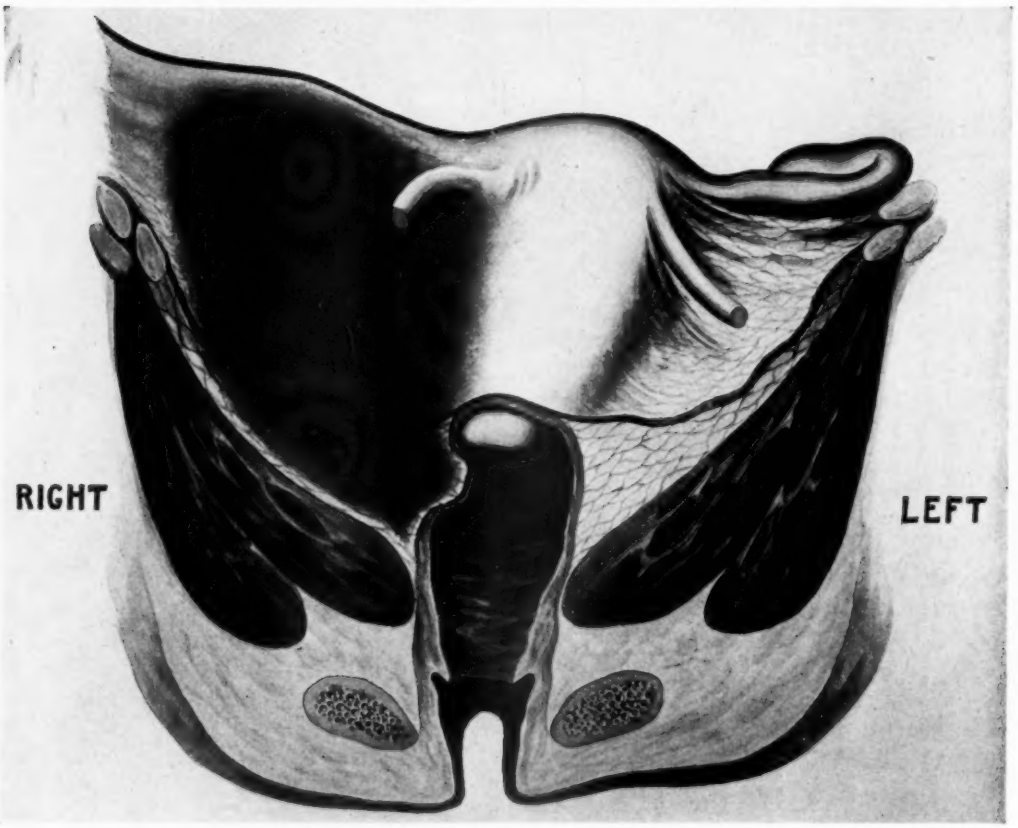


FIG. 22. Type II postabortal infection; exudate extending upward on the right side above the anterior superior spine and downward so as to bulge into the vagina. Extraperitoneal drainage could be established either above Poupart's ligament or below through the cul de sac; infection and drainage tracts all being extraperitoneal. (*Modified after Polak.*)

outward through the large veins into the broad ligament; or else it occurs secondary to the cellulitis in the broad ligament. The infection surrounding the walls of the blood vessels causes an arteritis with a slowing of the blood stream followed by changes in the endothelial lining with ultimate formation of a thrombus which becomes infected and the formation of a thrombophlebitis (Fig. 23). Occasionally fragments of this may break off and circulate in the blood stream as septic

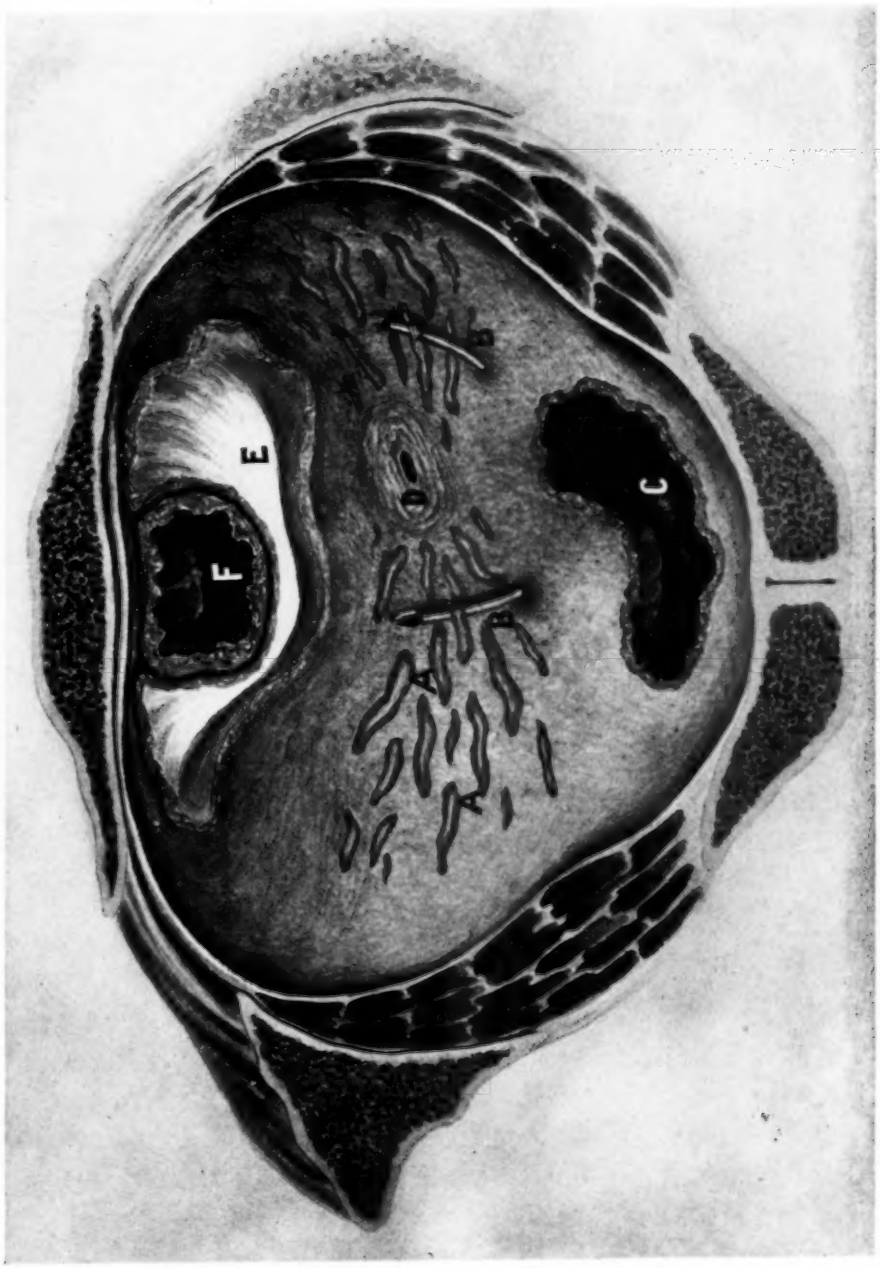


FIG. 23. Type III postabortal infection; transverse section of the pelvis showing thrombophlebitis in the veins of the broad ligament; also shows the exudate of the accompanying pelvic cellulitis.

emboli which cause localized areas of infection throughout the body, (occurred in one case in our series) and gives a positive blood culture.

FIG. 24. Type IV postabortal infection; showing A, localized abscesses in the wall of the uterus.

TYPE IV. ABSCESS OF WALL OF UTERUS. The infection may, in rare cases, form localized abscesses in the wall of the uterus (Fig. 24). Only one case occurred in our series.

Type III and Type IV rarely occur independently, they are usually associated with Type II. They usually cause a definitely recognizable group of symptoms.

TYPE V. GENERAL SEPSIS. This is a very virulent form of infection. In this type the extragenital symptoms may mask the etiological picture so completely that it is not recognized and the patient is not admitted to the Gynecological Service.

The organisms leave the uterus rapidly and localize in various organs so that the patient is admitted to the hospital with a diagnosis of pulmonary abscess, pneumonia or sepsis. These patients usually die and at autopsy it may be almost impossible to diagnose the condition as a postabortal infection. The uterine cavity may show no evidence of pregnancy or of any serious infection. *The more virulent the infection the less the reaction in the uterus.*

There are three paths by which the infection may travel from the uterus (Fig. 25):

1. Direct extension into the general circulation through the veins without injuring them, (no thrombosis);
2. By extension along the retroperitoneal lymphatics, the infecting organisms being carried directly into the subclavian vein from the thoracic duct; and
3. Retroperitoneal erysipelas. This form probably is the sepsis foudroyant of the French authors, one of the most virulent types of infection.

In all forms there is a blood stream infection with or without a positive blood culture. These organisms may become lodged in the various organs with the development of an endocarditis, bronchopneumonia, abscess of the lung, empyema, infarction of the spleen or kidney. In this type of infection the patients do not have a generalized peritonitis, the peritoneum is always clean.

Type VI. Generalized Peritonitis. These patients usually have a badly infected uterus with or without a gangrenous endometritis. The infection extends from the cavity of the uterus along the lumen of the tubes outward into the peritoneal cavity. The pus is spilled directly into the peritoneal cavity with the development of a generalized peritonitis (Fig. 26). For a long time this was considered as the only method by which a generalized peritonitis could develop. However, the possibility that in a Type II form of infection, in which there is a marked exudate in the broad ligament directly under the peritoneum causing a generalized peritonitis, must be allowed. It could occur either by direct extension through the lymphatics of the peritoneum, or the rupture of a small bleb, or of an abscess

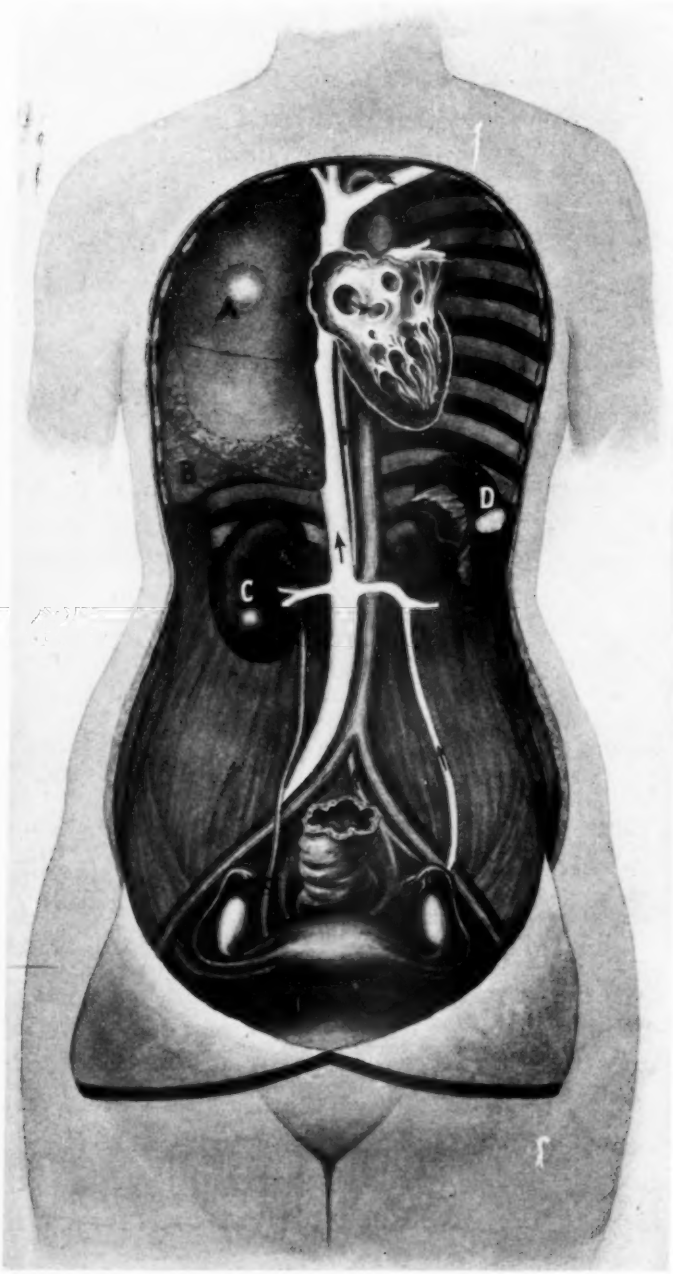


FIG. 25. Type V postabortal infection, general sepsis with a normal pelvis. The arrow in the ovarian vein shows the path of infection through the blood stream, without thrombophlebitis; arrow in the lymph tract (between branches of the iliac artery) shows the path through the lymph spaces to the thoracic duct directly into the subclavian vein; arrow in the heart shows infection on the endocardium and the valves; A, lung abscess; B, empyema; C, infarct in the kidney; D, infarct in the spleen.

into the peritoneal cavity. The lymphatic extension form is admitted on theoretical grounds only, as it has been impossible to demonstrate it in any of the cases treated. All cases of

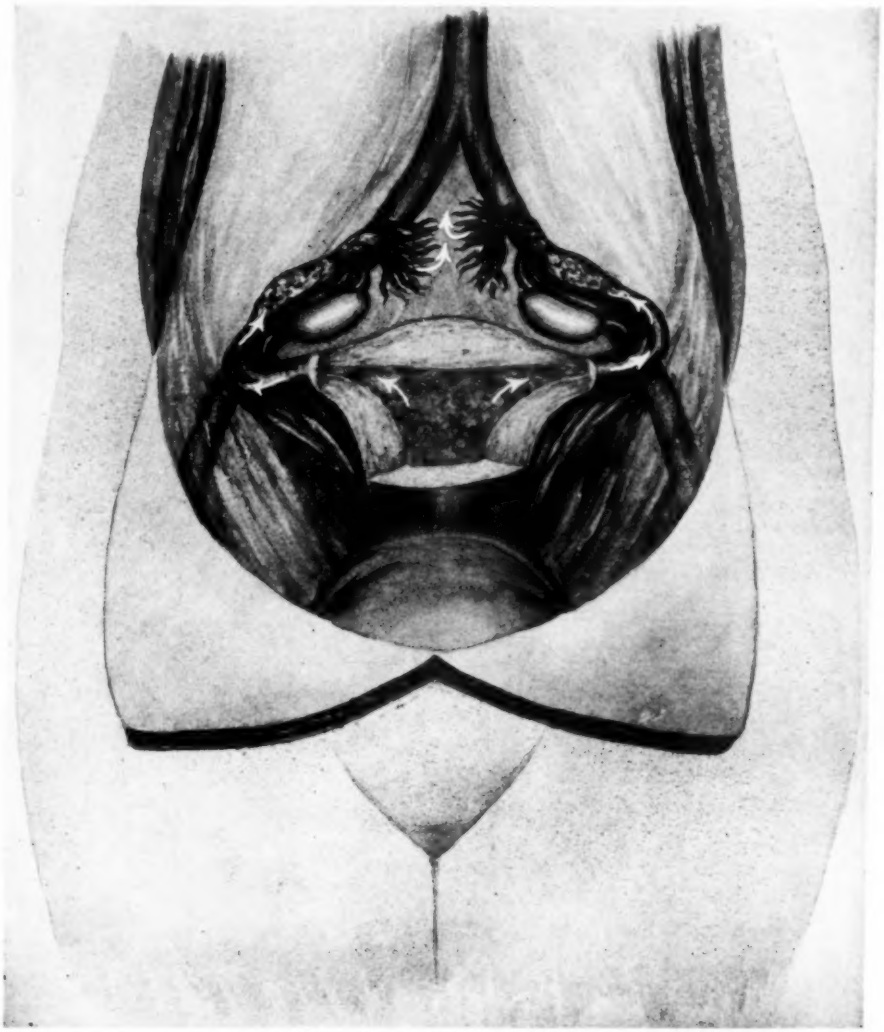


FIG. 26. Type VI postabortal infection; dark area inside uterus shows infected uterus (gangrenous endometritis); arrows through the tubes show the paths of the infection, gangrenous or streptococcic salpingitis, with direct extension into the peritoneal cavity and development of a general peritonitis.

peritonitis that occurred in this series resulted by direct extension through the tubes from a gangrenous endometritis, except in one instance which followed a rupture of an abscess.

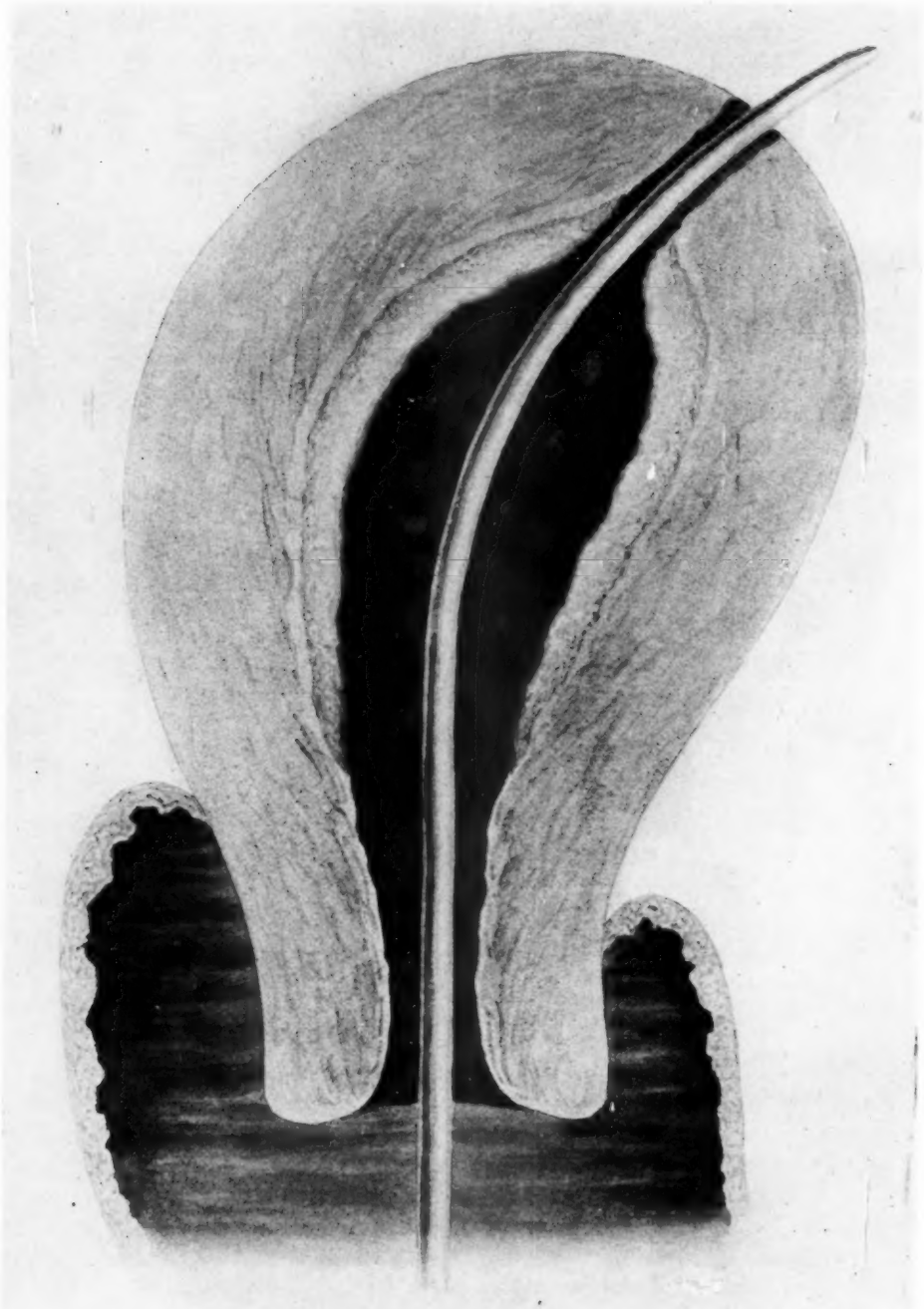


FIG. 27. Type VII postaboral infection; perforation of uterus showing sound going through uterine wall.

TYPE VII. PERFORATION OF THE UTERUS. This is not a true postabortal infection. When the uterus has been perforated in the course of a curettage, some viscus may be injured causing

FIG. 28. Three typical temperature charts of the Type I form of postabortal infection.

a general peritonitis (Fig. 27). The pathology depends largely upon the associated organs injured.

What is the clinical application of this classification and how can the type be recognized from the clinical picture?

The first two or three days after admission it is frequently very difficult to discern the type of infection present. The longer the patient lives, the longer she is under observation, the more accurate a pathological diagnosis may be made.

Type I. Infection limited to the uterine cavity, may or may not have a temperature. The height of the temperature (Fig. 28) is usually no criterion of the type of infection. However, if the temperature lasts for more than two weeks, the patient is rarely a pure Type I. Occasionally (five times in 1000 cases) a case is seen with infected uterine contents whose temperature lasts for more than two weeks and in whom no pathology is palpable in the broad ligaments (Fig. 29). With this exception the rule is that, *a patient who recovers and whose temperature did not last more than two weeks, is a Type I form of infection.*

Type II. Parametritis, etc. These patients have an irregular sustained temperature for more than two weeks. (Fig. 30.) The longest stay in the hospital in this type of case was nine

FIG. 29. Temperature chart of Type I postabortal case with infected uterine contents requiring surgical interference (sponge stick curettage).

months. When the sustained temperature in a Type II case begins to make wide daily excursion, 99° to 103°, one should suspect thrombophlebitis or the breaking down of a cellulitis with abscess formation. Surgery in the form of colpotomy, extraperitoneal drainage, etc., may then be indicated.

Do not operate on these patients transperitoneally. If these patients are operated through the abdomen, an extraperitoneal streptococcus infection is transformed into an intraperitoneal one with the development of a general peritonitis and death.

Type III. Thrombophlebitis. In Type II we have an irregular temperature which is moderately sustained. In Type III, however, there is a marked spiking of the temperature from 102° to 104° to normal (Fig. 31). When the sustained temperature of Type II begins to spike, think of thrombophlebitis or the localization of pus somewhere. Type III rarely occurs as a specific entity, it is usually associated with Type II.

Type IV. Abscess of the uterine wall, is a late complication of Type II. It rarely occurs alone. It usually occurs in the third, fourth or fifth weeks of the infection and causes a spiking of

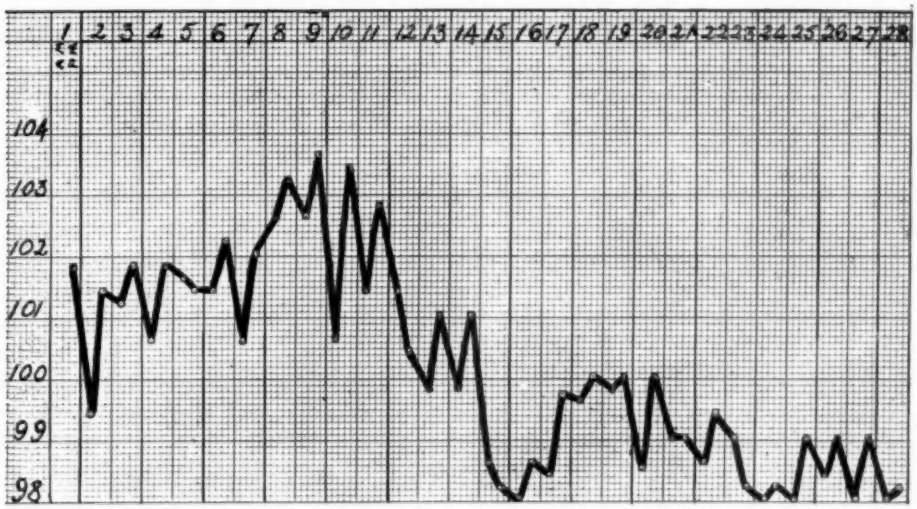


FIG. 30. Temperature chart of Type II form of postabortal infection; sustained temperature for twenty-four days. Physical examination showed the cervix to be fixed in a mass of exudate.

temperature similar to the localization of pus in the broad ligament.

Type V. General Sepsis. These patients live from one to six weeks. They have a septic temperature (Fig. 32) and are usually diagnosed as bronchopneumonia, empyema, meningitis or sepsis, unless one can elicit the abortion history. This type usually has a positive blood culture. Nearly all of these patients die in one to six weeks.

Type VI. General Peritonitis. This diagnosis, as a rule, cannot be made on admission unless one can obtain a very accurate history from a very intelligent patient. These patients present all the symptoms of a generalized peritonitis, rigidity, tenderness, leucocytosis, thoracic breathing, temperature (Fig. 33) etc. They usually die from seven to ten days after admission.

Type VII. Perforation of Uterus. The symptomatology varies according to the organ injured and how much gut has been pulled through the uterus, etc.

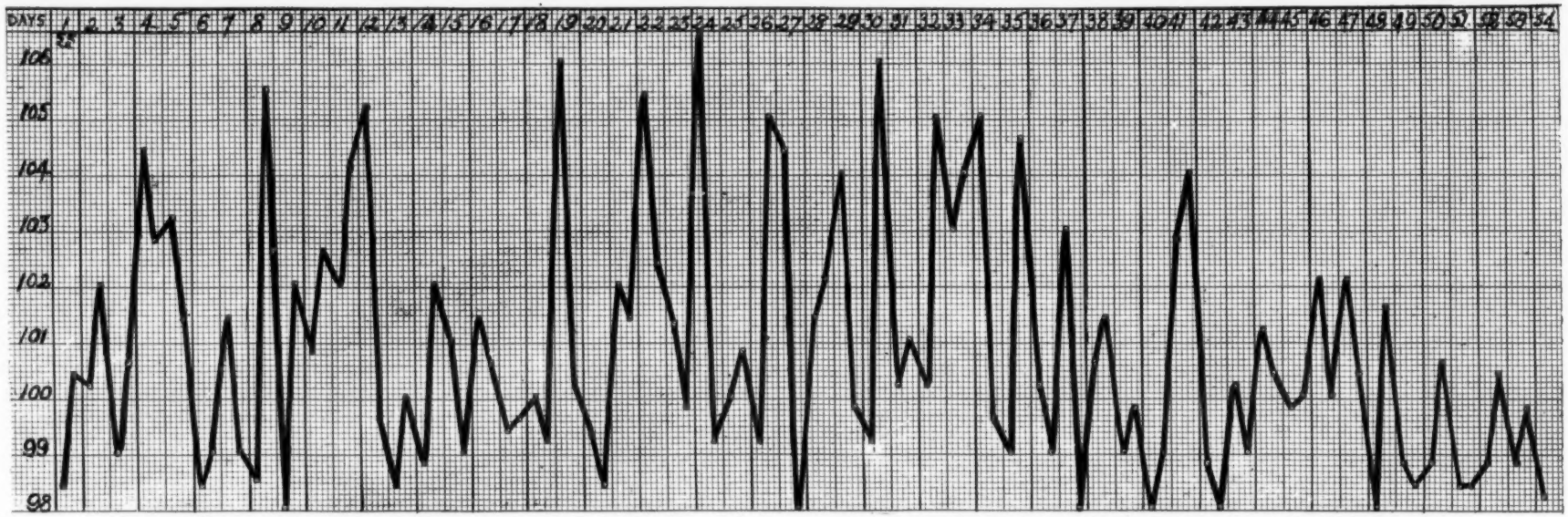


FIG. 31. Temperature chart of Type III postabortal infection and thrombophlebitis, with marked spiking of temperature for fifty-four days.

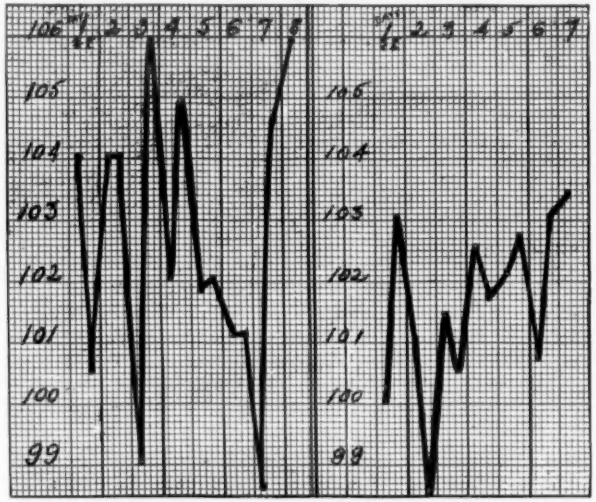


FIG. 32. Two temperature charts Type V form of postabortal infection; A, had positive blood culture, Streptococcus hemolyticus. Autopsy showed septic endocarditis, septic kidney and spleen, and gangrenous endometritis. B, had negative blood culture and was jaundiced on admission. Autopsy showed infarcts of lungs, kidney and spleen.

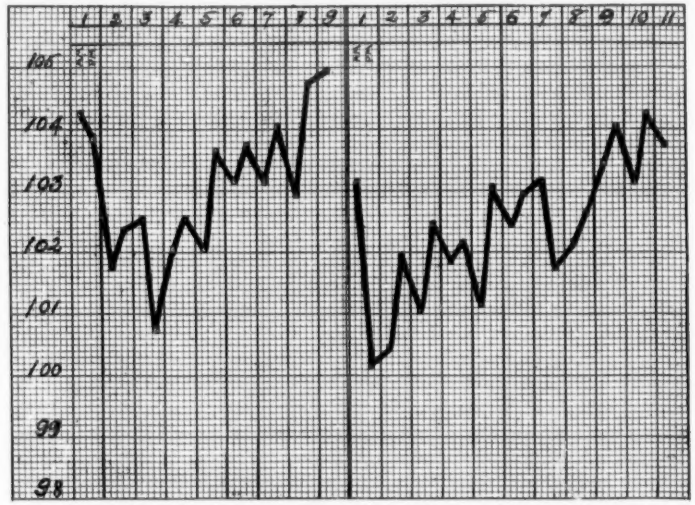


FIG. 33. Two temperature charts of Type VI form of postabortal infection. Both cases at autopsy showed gangrenous endometritis, gangrenous salpingitis and general peritonitis.

The Management of the Abortion

For therapeutic purposes only all abortions on admission are divided into two groups (this is not a pathological classification):

1. Threatened abortion
2. Incomplete abortion (which includes inevitable abortion)

A. Afebrile { 1. Without hemorrhage; 2. With hemorrhage

B. Febrile { 1. Without hemorrhage; 2. With hemorrhage

After taking a history, blood count, blood pressure and making a complete physical examination, the case is classified for therapeutic purposes. No attempt is made to make a pathological classification on admission. The exact pathological diagnosis is not made until the patient has been observed for several days. The longer a patient is under observation, the more accurate a pathological diagnosis can be made.

1. Routine for Threatened Abortion. If on admission the patient gives a history of a typical threatened abortion, e.g. she is pregnant, may or may not be bleeding or have cramps, has passed no tissue and the external os is closed, what shall be done for her?

She is put to bed and given opiates, codiene sulphate grain 1 every four hours by mouth, or morphine sulphate grain ¼ by hypodermic. She is shaved, an ice bag is applied to the abdomen and the patient is left undisturbed. *No vaginal examinations* are to be done except by the attending surgeon. *No cathartics* or *enemata are to be given.*

If the bleeding ceases and the symptoms clear up, the patient goes on with her pregnancy. If rest and sedatives fail, the case then enters the second group, incomplete abortion with or without hemorrhage and the necessary routine is carried out.

2*A*-1 Routine for Incomplete Abortion Afebrile without Hemorrhage. If on admission a diagnosis of incom-

plete abortion is made, the patient's temperature is normal and she has little or no hemorrhage, she is taken immediately to the examining room. The vulva is shaved and washed thor-

FIG. 34. Little's tube for intrauterine culture; A, small tube which may be glass throughout or may be of rubber with a capillary glass tube at its uterine end; B, outer glass sheath.

oughly with soap and water and flushed with a sterile solution (saline or bichloride). The parts are thoroughly dried. The thighs, lower abdomen, vulva and perineum are painted with a 4 per cent aqueous mercurochrome, or a 2 per cent mercurochrome in acetone and alcohol solution. The patient is draped with two sterile towels in preparation for the vaginal examination.

The examiner prepares himself as for an operation: scrubs up, gown, etc. A sterile speculum is inserted into the vagina and such blood as is found is wiped away. The vagina is flushed with the mercurochrome solution. This can be done with mercurochrome on a sponge wiping out the vagina thoroughly, or by spraying the mercurochrome into the vagina using a syringe. The excess of mercurochrome is wiped away and an intrauterine culture may now be taken if desired.

In making the culture it is very essential that an intrauterine and not a vaginal culture be made. For this purpose a Little's tube is used (Fig. 34). This consists of a tube within a tube. The outer tube is introduced through the cervix until it reaches above the internal os, the inner tube is then pushed through this tube into the uterine cavity. Suction is applied and the secretions from the uterus are drawn into the inner tube. The latter (the inner tube) is then withdrawn and its contents cultured.

Recently a group of Russians have developed a technique which seems to give even better results. They use an Ultzmann urethral cannula with a condom stretched over it. This is introduced through the cervix into the uterine cavity. A wire

is pushed through the cannula puncturing the condom which, being under tension, retracts. The wire is withdrawn and a syringe applied to the cannula. The uterine contents are drawn into the syringe and cultured.

Intrauterine cultures were taken routinely in 50 consecutive Type 2*A*-1 cases, (cases without temperature and little or no bleeding) to see if it would give any lead as to the future pathological course or therapy in any given case. It was found to be of academic interest only and did not help in any manner in regard to future course or therapeusis.

After a culture is taken, the vagina is again flushed with mercurochrome, the speculum is removed and a very gentle vaginal examination is made and the findings noted. *Gentleness in this procedure cannot be overemphasized.* At the autopsy table one can readily demonstrate how infected material in the lymph spaces can be dislodged by a rough vaginal examination.

The patient is put to bed in low Fowler's position. Ergot dram 1 every four hours is given for eight doses. *Ergot is never given without a stop order.* At the discretion of the examiner, pituitrin or gynergen may be given hypodermically. A mild cathartic such as milk or magnesia or mineral oil is given or a soap-suds enema is ordered. In three to five days if her temperature is flat and she has stopped bleeding, the patient is allowed out of bed, and is discharged on the fifth to the seventh day after admission.

2*A*-2 Routine for Afebrile Incomplete Abortion, with Moderate or Profuse Hemorrhage. The same procedure is carried out as for the afebrile abortion without hemorrhage, up to the introduction of the speculum.

Following the introduction of the speculum all blood clots and debris are removed from the vagina. The vagina is flushed with mercurochrome and *with another sterile sponge stick,* all blood clots and placental tissue in the cervix are removed. *Under no circumstances is the instrument to be placed above the internal os of the cervix.* If necessary, the cervix may be grasped with a sponge stick to steady it. If possible, avoid using a

tenaculum. If a tenaculum is used on the cervix, a note must be made on the chart for medicolegal purposes.

After cleansing the cervix, the vagina is again flushed with mercurochrome and at this point a uterine culture can be taken. If all placental tissue has been removed the patient will stop bleeding. If the bleeding has practically ceased, the speculum is removed. A gentle vaginal examination is made, the findings are recorded and the patient is then treated as Group 2*A*-1 (afebrile case with little or no hemorrhage). If the bleeding has not been controlled, no vaginal examination is made at this time but the vagina is thoroughly packed. *The uterus is not packed.* The packing is introduced up to the internal os of the cervix. If the vagina is properly packed, the patient should not be able to void. *Iodoform gauze should never be used.* Three cases of vaginal stricture with almost complete obliteration of the vagina following iodoform vaginal packing have been observed. Plain packing soaked with mercurochrome should be used. As the packing is put in, mercurochrome is sprayed on the packing with a syringe. The patient is returned to bed and *across the front of her chart is placed a piece of adhesive* marked with the date and hour when the packing took place and the number of pieces of packing in the vagina. This adhesive should not be removed except by the individual who removes the packing. When the packing is removed a note is made on the patient's chart and signed by the individual who removed it. Then and only then, is the adhesive removed from the front of the chart. If this precaution is not taken, patients will return to the follow-up clinic with a putrid, vaginal discharge and on examination a piece of gauze will be found in the vagina.

After the patient is packed, she is placed in low Fowler's position, given fluids by mouth, a soft diet, a daily enema or a mild cathartic like milk of magnesia or mineral oil. All these patients are typed for transfusion. They are given a clysis, infusion or a transfusion at this time, to make up for blood loss, e.g., loss of fluid volume only. Patients are admitted who are exsanguinated from an incomplete afebrile abortion. As a

result of the tremendous loss of blood volume, they appear to be in shock. The transfusion of 500 c.c. or more at this time is a therapeutic measure to combat shock and hemorrhage and not infection.

Medically the patient is given fluid extract of ergot dr. 1 every four hours for eight doses and if necessary gynergen or pituitrin hypodermically. Sedatives may be given, when indicated.

The primary packing is removed within twenty-four hours and if the bleeding is not controlled, repacking may be necessary. Each time the patient is repacked, she and the operator are to be prepared the same as on her first admission. Three packings may be done with impunity. *The number of packings that a patient will receive are in proportion to the care with which the first packing is made.* Usually one packing is sufficient, occasionally two are necessary. If three or more are necessary, someone is "falling down on the job."

Should a temperature develop following any packing, it is immediately removed and the patient is treated as a febrile incomplete abortion.

If the patient has been afebrile without bleeding for five days after removal of the packing, she is allowed out of bed and discharged on the seventh day.

2*B*-1 ROUTINE FOR INCOMPLETE ABORTION WITH FEVER AND WITHOUT HEMORRHAGE. The preliminary routine is exactly the same as in the afebrile incomplete abortion with moderate or profuse hemorrhage, up to the point where the packing is introduced.

After the regular vaginal and cervical cleansing, the patient is put to bed in low Fowler's position unless contraindicated by shock or marked loss of blood. Fluids are given by mouth and the bowels are kept open by mild catharsis or daily enemata. A blood culture is routinely taken in every case of abortion with a temperature over 102°. The blood culture is repeated every other day until a positive culture is obtained or the clinical course of the patient rules out a blood stream infection.

Medically the patient receives fluid extract of ergot dram every four hours for eight doses, gynergen or pituitrin by hypodermic if indicated and a sedative if necessary. A clysis, infusion or transfusion, for replenishing fluids may be given on admission.

2*B*-2 Routine for Incomplete Abortion with Fever and Moderate or Severe Hemorrhage. The preliminary routine is exactly the same as afebrile incomplete abortion with moderate or profuse hemorrhage, with the exception that vaginal packing is used only where there is absolutely no possibility of controlling the hemorrhage by any other means. When packing is used, it is removed after twelve hours and not repeated except as a life-saving measure. The immediate further management is the same for incomplete febrile abortion without hemorrhage. Blood cultures are taken in the same manner as in febrile abortions without hemorrhage.

The majority of patients can be discharged within two weeks. Pathologically they fit into the Group 1 type of postabortal infection and on discharge are classified as "Pregnancy, abortion completed."

Those patients that are not discharged within this time and who remain on the wards as a result of continued bleeding or temperature, are then reclassified from the pathological standpoint and their further treatment is guided by the pathology found. The pathological diagnosis is made on the basis of the history, clinical course, temperature, pulse, physical findings, etc.

Management of the Cases Which Continue to Bleed. If a patient continues to bleed moderately, if she is afebrile for some time, has a patent cervix and there is nothing palpable in the adnexa, that patient has something within the uterus that is causing the persistent bleeding. These cases fall into Type 1 postabortal infection, e.g., those cases where the infection and the infected material is limited to the cavity of the uterus and has not extended beyond it into the broad ligaments.

These patients are taken to the operating room and the uterus is entered under strictest asepsis and antisepsis. It is

wiped out with gauze soaked in 2 per cent mercurochrome, acetone-alcohol solution or 7 per cent tincture of iodine. A sponge stick is introduced into the uterus and all free tissue is removed. The uterus is again wiped out with iodine or 2 per cent mercurochrome solution and the patient returned to the ward.

Management of the Cases with Continued Temperature. All cases with continued temperature are reclassified either as infected abortion or septic abortion. Owing to the difficulty in obtaining a true history, every abortion admitted to the hospital is regarded as an induced abortion and therefore potentially infected. The differentiation between an infected abortion and a septic abortion is a purely clinical differentiation for therapeutic purposes only. The differential diagnosis is occasionally quite difficult. This can be readily understood if one realizes that all septic abortions are infected abortions, but all infected abortions are not septic. The specific line of demarcation when an infected abortion enters the septic group is purely arbitrary. If one relies upon positive blood cultures only as the differential, it will be found not infrequently that a patient will die with all the symptoms, temperature variations, pulse changes, etc. of a septic abortion without a positive blood culture.

Those cases of abortion are regarded as infected where the invasion and growth of microorganisms are limited to the uterus and the toxins only act on the general organism. When the pathogenic organisms have left the uterus and entered the tissues of the body locally or generally, those cases are classified as septic.

In studying a series of 1000 consecutive abortions it was found that patients almost automatically separate themselves into the infected and septic groups. Those cases where

1. The temperature lasts for less than two weeks irrespective of type or degree;
2. The blood cultures are all negative;
3. No peritonitis develops; and
4. No extrauterine masses or pathology can be demonstrated,

are clinically infected abortions. They always recover. The infection is limited to the uterine cavity.

Those cases where

1. The temperature is above 100.4° for more than two weeks;
2. A positive blood culture is obtained;
3. Symptoms of parametritis with pelvic masses develop; and
4. Peritonitis develops,

are septic abortions. These symptoms may develop before two weeks and the diagnosis of septic abortion made at that time. The diagnosis of septic abortion however, is very rarely made on admission and then only when an absolutely reliable history justifies it.

All infected abortions are potentially septic cases and must be handled and regarded as such. All patients with infected abortions must be regarded as seriously ill cases and must be given a considerable amount of personal attention.

Management of the Septic Abortion. Following the admission, the febrile incomplete abortion routine has been carried out. *If the temperature continues for more than two weeks, if masses are felt in the pelvis or if a positive blood culture is obtained, the case is regarded as septic.*

This is true in practically all cases. Only five exceptions were found in a series of 1000 cases where the patient's temperature remained above 100.4° for more than fourteen days, the uterus remained enlarged and the external os patent. Vaginal examination ruled out any tender areas or masses in the cul-de-sac or fornices and a diagnosis of *infected uterine contents with poor drainage was made.* A sponge stick was introduced into the uterus with strictest asepsis and antisepsis. In handling these cases of abortion mercurochrome and iodine solutions are used very freely. The plug in the uterine cavity which was obstructing its drainage was removed so as to allow free drainage; and these patients all made an uneventful recovery.

As soon as the case is classified as a septic abortion, *no vaginal manipulations of any kind* are made unless there is a question of a localizing abscess. If the patient's temperature, which has been maintaining a plateau, begins a daily range

from 99° up to 102° or 103°, one should immediately suspect the possibility of a softening or localization of the exudate, with the formation of an abscess. In these cases a very gentle vaginal examination is to be made. If drainage is indicated, surgical intervention such as posterior colpotomy or extraperitoneal drainage may be carried out.

Medically supportive treatment and good individual nursing are essential. The patient must receive 3000 c.c. of fluid daily. A record sheet with the fluid intake and output should be kept on every case of septic abortion. She should receive a minimum of 2000 calories a day in her diet; and when the diet falls off due to lack of appetite, insulin given regularly generally helps very materially. The insulin should be given starting with two units t.i.d., a.c. and gradually increasing up to ten units t.i.d., a.c. The use of insulin in this way, causes a marked increase in appetite and forces the patient to accept a sufficient diet with the necessary calories. With the use of insulin and a measured caloric diet it has been possible to discharge patients weighing five pounds more than on admission, in spite of a septic abortion with a sustained temperature for eight months.

The value of repeated small transfusions in septic abortions of 150 to 250 c.c. of blood every third or fourth day as long as necessary, cannot be overestimated. Transfusions should not be begun unless one is able to continue giving them. One large transfusion of 500 c.c. or more is not as valuable as the repeated small ones. It is apparently the stimulating effect of the blood that helps as much as the blood volume.

Good nursing, fresh air, e.g. porch treatment when it can be carried out, are very helpful. Symptomatic treatment, surgical or medical, is carried out for such conditions as may develop.

Surgical Management of the Septic Abortion

When the exudate (parametritis) localizes or softens with the formation of an abscess, a colpotomy (vaginal incision) is indicated.

Colpotomy is not a new procedure. Whether or not one should wait until the abscess develops or incise the indurated area before softening, has been a point of contention since 1877. In 1877 Dr. Warren Brickell stated,

> My conviction is that if early operative procedure is once admitted to be proper (and for this I strenuously contend), the operation per vaginam will take complete precedence. I am satisfied that it must be rarely exceptional to find the purulent collection early "pointing" or inviting the knife or trocar towards the external surface. Long before this stage and when the abscess is comparatively small, puncture per vaginam will promptly relieve. There is a positive demand to remove serum as to remove pus; nevertheless it would be almost a hopeless task to convince any considerable number of surgeons to resort to such practice without practical illustrations to support the theory.

This is just as true today as when it was written. It is still a moot question as to whether or not postabortal masses should be drained before fluctuation develops.

There are two methods of doing a posterior colpotomy:

1. The patient is placed in the lithotomy position. The vagina is painted with mercurochrome 2 per cent acetone-alcohol, the patient is catheterized and she is draped. A weighted speculum is inserted into the vagina and the posterior lip of the cervix is grasped with a long tenaculum forceps. The weighted speculum is pulled down, the cervix is drawn up by the tenaculum and a long needle of 8 or 10 gauge with syringe attached, is introduced into the small dimple behind the cervix in the direction of the mass for a distance of 1½ to 2 inches or until the needle apparently enters the pus sac. The piston of the syringe is withdrawn and if the needle is in the abscess cavity, pus is obtained. The needle is left in situ, the syringe is removed and the pus is immediately placed in a test tube for culture. With a knife, a small transverse incision is made to either side of the needle through the mucosa of the posterior vaginal wall. A long curved Mayo scissors is introduced along the needle into the abscess cavity. The blades of the scissors are separated and the scissors removed with the blades open, so as to leave a large opening in the vaginal vault. The finger is introduced into the cavity and all pockets are broken up.

A special drain T-tube is used. These can be bought ready made or can be made in the operating room. To make the drain, take a large, fairly soft rubber tube ¾ of an inch to 1 inch

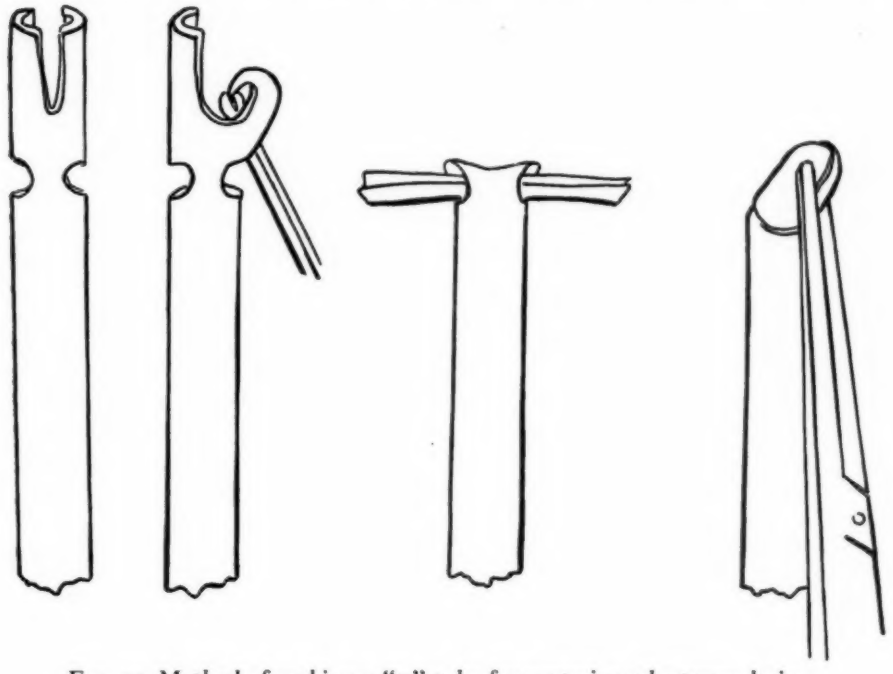


FIG. 35. Method of making a "T" tube for posterior colpotomy drain.

in diameter and at a distance of 1 inch from the end of the tube, cut two openings opposite each other (Fig. 35). Cut a wedge shaped piece from the tube on the two other sides down to the level of the middle of the openings. Pass an artery clamp through the opening, grasp the top of the tubing on the same side, and draw it through the opening. Do the same on the other side. This gives a fairly large opening for drainage and and also two shoulders to hold the tube in the opening in the posterior cul-de-sac.* This drain is inserted into the abscess cavity with a dressing forceps. A piece of gauze is placed around the tube. This gauze is removed in twenty-four hours. When the patient's temperature becomes normal, the tube is removed. If the temperature does not have a tendency to come down or if, after it has begun to come down, it again begins to go up, the

*FALK, H. C. Operating Room Procedures. New York, G. P. Putnam's Sons, 1934, p. 94.

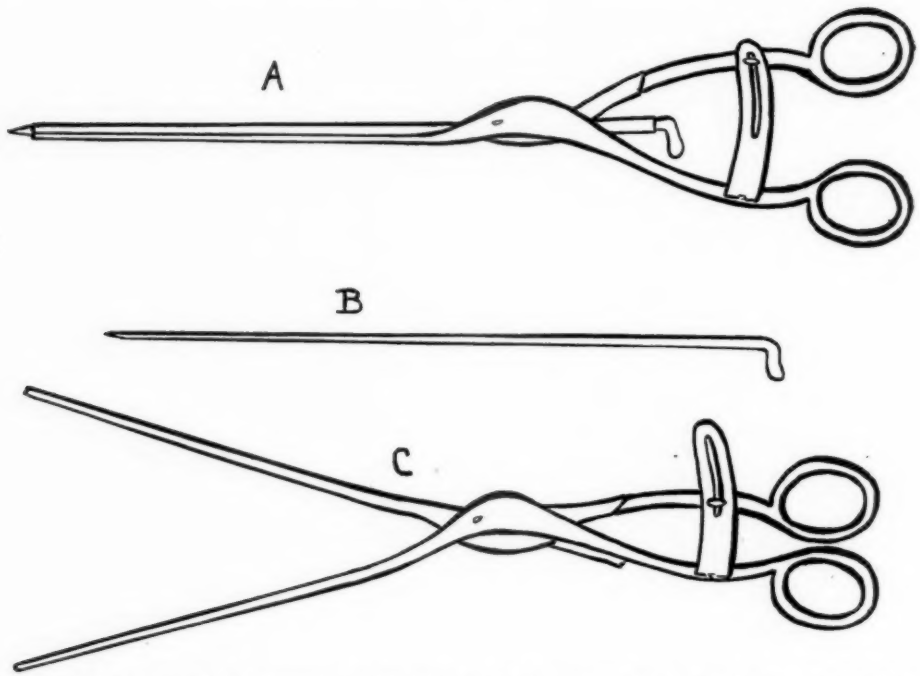


FIG. 36. The Fraenkel instrument showing A, trochar-cannula; B, trochar; C, spreading blades.

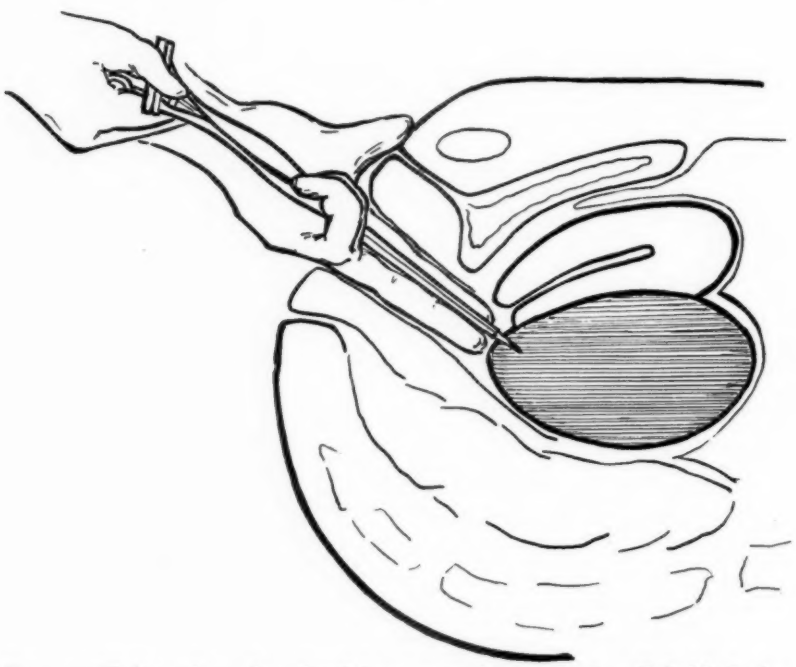


FIG. 37. Point of trochar fixed into posterior vaginal wall behind the cervix.

tube is removed to allow freer drainage. These tubes may become clogged. After the removal of the tube, the finger is introduced into the opening in the vault of the vagina daily

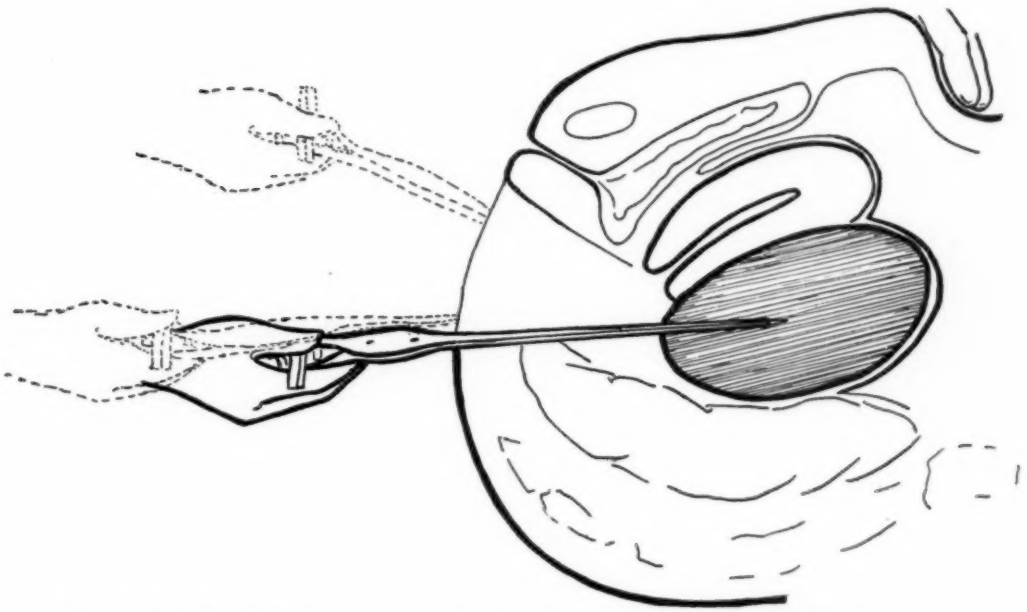


FIG. 38. Handle of the instrument is depressed toward the rectum and the instrument pushed forward towards the center of the mass, using the hand on the abdomen as a guide. The trochar is withdrawn and if pus flows through the cannula it is cultured.

so as to keep the colpotomy wound open.

2. Fraenkel technique, using the Fraenkel instrument. This consists of a sharp pointed trochar cannula and a glove stretcher dilator (Fig. 36). The patient is placed in the lithotomy position. The usual vaginal preparation is carried out. The patient is catheterized, painted and draped. Two fingers are introduced into the vagina and the cervix is palpated. The Fraenkel instrument is introduced in front of the fingers until the point of the instrument is fixed in the posterior vaginal wall, at a point 1 to 2 cm. behind the posterior lip of the cervix (Fig. 37). The handle of the instrument is then depressed toward the rectum (Fig. 38) so that the trochar cannula points towards the exudate, which is usually close to or associated with the posterior wall of the uterus. The instrument is then pushed forward into the exudate or toward the mass using

the abdominal hand as a guide and the trochar is removed. If it is in a pus sac, pus will flow through the cannula. A culture of this pus is taken.* The handle of the instrument is closed,

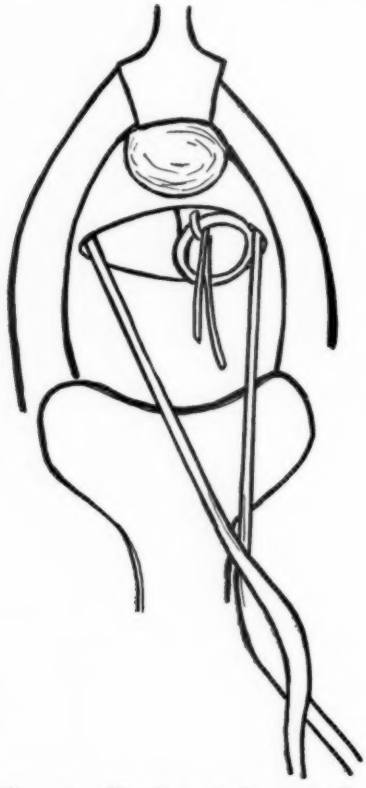


FIG. 39. Handle of the cannula closed, tearing a hole in the posterior vaginal vault. Hard rubber perforated drain introduced through the opening into the abscess cavity.

opening the glove stretcher dilator and tearing a large opening in the posterior vaginal wall (Fig. 39). A wedge shaped piece of hard rubber tubing is then introduced into this opening. This piece of tubing has openings on its side and there is a piece of gauze attached to its base, so that it can be removed (Fig. 40). This cone shaped tube is held in the opening by the concentric contraction of the vault of the vagina. The tube

* FALK, H. C. Posterior colpotomy for pelvic infection. *Am. Jour. Surg.*, 9: 494, 1930.

acts like a keystone. A piece of packing is introduced into the vagina and the patient is returned to the ward. This hard rubber tube is allowed to remain in place ten days and any

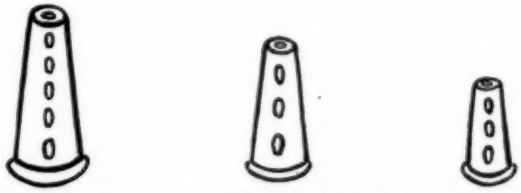


FIG. 40. Three sizes of hard rubber drainage tubes with lateral perforations.

form of vaginal heat treatment that is desired may be followed, as the end of the tube does not project into the vagina. These patients, if afebrile, may be allowed up and around with the tube in place. They are discharged about the tenth day. Occasionally they have been allowed to go home with the tubes in situ and have returned for treatments, the tube being removed on the twelfth or sixteenth day.

If the patient's temperature does not subside or if it rises after having been down, one should think of the possibility of the formation of another abscess in which case another colpotomy may be necessary.

Occasionally the pus or exudate forms in front of the uterus towards the bladder. When this happens an anterior colpotomy may be necessary. The same procedure is followed but a drainage tube is not introduced as it might slough into the bladder. In doing an anterior colpotomy one must be absolutely sure that the bladder is empty just before introducing the needle or the Frankel instrument.

In occasional cases the exudate goes laterally and upward reaching above the iliopectineal line, extending along the iliac fossa and appearing as a mass above Poupart's ligament, the anterior superior spine or the crest of the ilium (Fig. 22). These cases may require an external incision for extraperitoneal drainage. The patient is prepared as for a laparotomy. The incision is made a finger's breadth above and parallel to Poupart's ligament. The external oblique fascia is exposed and

split in the direction of its fibers retracting the edges of the opening. The internal oblique is thus exposed and is lifted upward. The fingers are introduced downward toward the ilium and along the iliacus muscle, lifting the transversalis fascia upward. The finger is introduced down to the iliopectineal line. Irrespective of the finding of pus or inflammatory exudate, four empty Penrose tubes are introduced and the wound is packed. The patient is returned to bed and given supportive treatment. At a later date the pus, following the path of least resistance, will drain extraperitoneally.

Question: In the absence of a positive blood culture would the case be classified as a septic abortion?

Answer: Yes. The term septic is a purely clinical one and any patient in whom the infection has left the uterus and involves other tissues or organs is regarded as septic. This classification is essential especially from the standpoint of therapy.